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MEASURING THE RISK OF INFECTION AT WORK

BY

DAVID HEWITT and ALICE STEWART

From the Institute of Social Medicine, Oxford

The present investigation was prompted by the Northamptonshire Miniature Mass Radiography Survey of 1945-46. One of the findings of this survey, as presented by Smith (1947), was a significantly higher proportion of newly-discovered cases of active pulmonary tuberculosis* among boot and shoe factory workers than among workers in other trades. In July, 1947, the manufacturers asked for an investigation to be carried out, with the object of discovering whether this higher incidence of tuberculosis was the result of working conditions, or whether it was due to factors outside the control of the industry. Since then five related papers have been published. Stewart and Hughes (1949, 1951) describe how an intensive scrutiny of the mass radiography records broke down the total of cases found in boot and shoe factories into a number of homogeneous groups, and, notably, how it was possible to discern an association between cases of chronic tuberculosis and newly-infected cases. The theory that selective recruitment is solely responsible for the high tuberculosis morbidity rate is criticized, and attention drawn to the much higher rates prevalent in large than in small factories. An experiment carried out in Northampton and Leicester by Hirch (1951) shows the level of bacterial contamination of the air in boot and shoe factory workshops to be largely determined by the number of persons present, however close together or far apart they may be. Cairns and Stewart (1951) present a comparative history of the printing and shoe-making trades, to show that, while the living standards enjoyed by shoemakers are not such as would be expected to cause an excess of tuberculosis, there is some plausibility in the theory of a long-wave epidemic in the factory section of the shoe-making community. Webb, Stewart, and Sutherland (1951) discuss the tendency, revealed in a street survey of Northampton, for cases of tuberculosis to occur in adjacent houses. These papers all tend to strengthen the view that infection outside the home is an important factor in the tuberculosis morbidity of boot and shoe workers.

Since the original mass radiography survey, fresh material has been gathered which forms the basis of this paper. A further attempt is now made to trace the path of infection in boot and shoe factories, to suggest appropriate measures for

* Throughout this paper " tuberculosis " is to be understood as " pulmonary tuberculosis ".

reducing the disease incidence, and to draw some conclusions which may be of general epidemiological interest.

(1) Material Used in the Present Study

The new material comprises:

(*a*) Records of all boot and shoe factory operatives notified as cases of tuberculosis in the Borough and County of Northampton during the decade 1940-49. There were nearly 600 such records, each including the occupation and last place of employment before notification. The placing of cases in individual workshops (see Table I, opposite) is based on this information.

(*b*) Board of Trade returns from 194 boot and shoe factories in the same area, showing the number of workers employed on production in each of the years 1941-1949. These include all the factories in continuous production during the period of study.

(*c*) The results of a survey of the same factories made in 1948 to determine the number of men and women working in each room or compartment, distinguishing the six main occupational groups.

(*d*) For a smaller number of factories*, approximately two-thirds of the whole, the floor area of each room or compartment was also obtained. From these measurements the floor space per worker was calculated (see Table II, p. 212).

Compared with the mass radiography survey, the present body of data is more comprehensive but less clear cut. It is, for instance, less certain that the last place of work before notification was the place at which the worker first began to develop tuberculosis. With the mass radiography data there was no difficulty in establishing comparable morbidity rates for different sections of the population examined. In the present instance, so-called notification rates have been calculated from the total number of cases and the number of persons at work in 1948, but these rates are only comparable one with another if the relationship between the 1948 working population and the total exposure is the same for each group examined. Fortunately, such a relationship may be assumed, since the processes involved and the machinery used prescribe very nearly standard working conditions for all the factories. The history of most of the factories was also very similar throughout the decade.† While, therefore, the lack of precision in the data may have blurred the outlines of the main pattern, there is no reason to suppose that it has caused distortion.

(2) Units of Study

Three basic groupings of workers have been studied:

(*a*) factories; (*b*) walled rooms; (*c*) compartments‡.

* This sample had the same age, sex and occupational composition as the whole of the material and included factories of all sizes. There was, however, a tendency for the measurements to be obtained more frequently from the larger factories.

† Of the 194 factories covered by the Board of Trade returns, 17 were " concentrated " during the war. These have been excluded from the calculations. A further 20 subsidiary factories which were included in the mass radiography survey but which did not carry out the complete manufacturing processes were also excluded. The remaining 177 factories accounted for 515 persons notified during the decade 1940-49.

‡ A compartment is co-extensive with a walled room where there is no subdivision of the room. Wherever a permanent partition was found which reached eye level but did not touch the ceiling, it was held that the workers on either side belonged to separate compartments.

The obvious advantage of the factories as units of study was that their notification rates, being based on relatively large numbers, were much less erratic than the corresponding rates for the other two groupings. There are, however, in factory populations, significant variations associated with age, sex, and occupation, which influence the liability to develop tuberculosis. Since all the factories included in the survey had approximately the same proportion of workers of each sex and occupation, these variations would inevitably have been overlooked, had factories been made the basis of study. Compared with the walled rooms, the compartments more often contained members of only one sex or only one occupational group. It was decided, therefore, that from the point of view of homogeneity they were better than either of the other groupings.

(3) Effect of Partitions

Since partitions have some of the characteristics of walls, it is of interest to compare the notification rates in partitioned and unpartitioned rooms. This point is examined in Table I, where, for each of three size groups, the notification rates for the two types of room are compared. In each group the difference between the frequency of tuberculosis in rooms with and without partitions is statistically significant, the lower rates occurring always in the partitioned rooms. This finding suggests that the presence of partitions has had an appreciable effect on the risk of cross-infection at work and is a further reason for preferring compartments as the basic units of study.

TABLE I

Effect of Partitions on Frequency of Tuberculosis

Type of Room		No. of such Rooms	No. of Workers, 1948	Cases of Pulmonary Tuberculosis, 1940–49	Notification Rate, 1940–49	Average No. of Workers per Room, 1948	Rate in Partitioned Rooms lower by (per cent.)
No. of workers	Partitions						
1 to 75	None	502	10,590	183	1·73	21·1	31
	One or more	54	1,854	22	1·19	34·3	
76 to 150	None	35	3,681	101	2·74	105·2	38
	One or more	26	2,722	46	1·69	104·7	
151 or more	None	7	1,555	48	3·09	222·1	7
	One or more	18	4,026	115	2·86	223·7	
All Rooms		642	24,428	515	2·11	38·1	

The rooms were divided into three groups according to the number of workers they contained. For each group a χ^2 test of the hypothesis that risk of tuberculosis is not affected by partitioning was performed. The combined results of these tests give a value of χ^2 equal to 10·54 with three degrees of freedom, the corresponding value for P being less than 0·02. In the first size group it so happened that the rooms with partitions were

of greater average size than those without. Hence the 31 per cent. reduction in the notification rate is likely to be an underestimate of the effect of partitions. On the other hand, the reduction of 7 per cent. in rooms with over 150 workers could not by itself be held to have proved a real difference between the two types of room.

(4) Effect of Spacing

It is a familiar notion that overcrowding, both by multiplying opportunities for infection and by lowering hygienic standards, increases the risk of contracting infectious diseases. Tuberculosis in particular, since it is thought to require close and sustained contact for its passage from one person to another, is spoken of as a disease of overcrowding. We expected, therefore, to find the highest notification rates in the most congested working units, but this expectation was not fulfilled (see Table II).

TABLE II

Apparent Association between Spacing of Workers and Frequency of Tuberculosis

Range of Area per Worker (sq. ft)	No. of Workers	No. of Notifications	Notification Rate (%)	Average No. of Workers per Compartment
0–55	6,279	140	2·23	54·6
56–85	4,344	102	2·35	33·7
86–120	4,681	128	2·73	36·9
121–175	3,126	76	2·43	25·4
Over 175	1,460	15	1·03	11·9

When the compartments were first classified by area-per-worker it seemed that the maximum risk of tuberculosis occurred in the middle of the range; i.e. units with more than 120 sq. ft per worker had notification rates below the maximum, *but so did those with less than 55 sq. ft.* This obvious paradox was resolved when it was realized that there was considerably more variation of area-per-worker from one occupational group to another than within each occupational group. The notification rate for units with less than 70 sq. ft per worker was in effect a rate for " closers "; in units with more than 150 sq. ft per worker there was a preponderance of " rough stuff " and " shoe room " workers, while the other occupational groups also fell within fairly well-defined ranges of area-per-worker. Since the occupational groups differ very widely in sex composition, they also differ in their liability to develop tuberculosis. It was decided, therefore, that the association between crowding and risk of infection should be studied separately in each occupational group (see Table III).

All units containing workers from more than one occupation were excluded, and the remainder divided into six occupational groups. Each group was in turn subdivided into as many area-per-worker groups as the material allowed. For each of the six occupational groups a χ^2 test was then performed for the hypothesis that, within each occupation, the distribution of cases of tuberculosis was independent of area-per-worker. The results of the tests were then pooled to obtain a value of χ^2 equal to 23·48 with 17 degrees of freedom, for which P is greater than 0·10. For the sake of contrast six more tests were

TABLE III

LACK OF GENUINE ASSOCIATION BETWEEN SPACING OF WORKERS AND FREQUENCY OF TUBERCULOSIS

Occupational Groups	Degrees of Freedom in χ^2 Test	Result of Testing whether Tuberculosis is Independent of			
		(*a*) Area per Worker		(*b*) Workers per Unit	
		χ^2	$P(\chi^2)$	χ^2	$P(\chi^2)$
Clickers	5	5·28	·50>*P*>·30	11·82	·05>*P*>·02
Closers	4	3·70	·50>*P*>·30	18·50	·001>*P*
Rough Stuff	1	9·17	·02>*P*>·01	0·53	·70>*P*>·50
Makers	3	2·78	·50>*P*>·30	4·80	·20>*P*>·10
Finishers	2	1·10	·70>*P*>·50	3·43	·20>*P*>·10
Shoe Room	2	1·45	·50>*P*>·30	8·17	·02>*P*>·01
All Occupations Combined	17	23·48	·20>*P*>·10	47·25	*P*<·001
All Occupations except Rough Stuff ..	16	14·21	·70>*P*>·50	46·72	*P*<·001

made for the hypothesis that, within the same restricted material, the distribution of cases was independent of the number of workers per unit. In this case the sum of χ^2 equals 47·25, for which P is less than 0·001.

These tests still show that there is no significant evidence of higher morbidity rates in the more congested working units. Since, in general, the compartments with most workers are also those in which area-per-worker is least (Table II, column 4), it is all the more remarkable that no significant area effect appears. It has to be concluded that spacing does not affect the risk of infection within the range of variation observed. Area-per-worker has therefore not been taken into consideration in the following analysis.

Some explanation should, however, be attempted for the special case of the "rough stuff" workers. For this one occupational group the χ^2 tests suggest that risk of infection may depend on area-per-worker, rather than on number of workers per unit. A possible explanation is that there may be some critical distance between workers, beyond which the benefit of wider spacing begins to be felt, and that only in the rough stuff rooms is this critical distance exceeded. On this view a crowding effect fails to appear, not because area-per-worker has no bearing on risk of infection, but because *boot and shoe factories as a whole are "overcrowded"**, in the sense that the distance between workers is nearly always less than some critical amount. The possibility that such a critical distance may exist will be discussed again in Section 5. Whether it exists or not, there are three possible advantages enjoyed by rough stuff workers which are worth mentioning:

(*a*) Available evidence suggests that known tuberculous subjects tend to avoid rough stuff work. If this is so, sources of infection will be much fewer than in the industry as a whole.

* This use of the term overcrowding should not be confused with "statutory overcrowding". In fact, the air-space per worker in most of the workshops far exceeded the minimum prescribed by law.

(*b*) The bulky machines and tall racks found in rough stuff work may act like the partitions already discussed.

(*c*) Bacterial counts per cu. ft of air recorded by Hirch (1951) were lower in the rough stuff departments than elsewhere.

It should also be mentioned that although rough stuff is the smallest of the six occupational groups examined, and too small for further subdivision, it is not homogeneous either in sex or in operation performed. The difference in risk which appears to be due to difference in spacing might disappear if it were possible to give separate treatment to the individual occupations grouped together as " rough stuff ".

(5) First Measurement of Risk Gradient

Having decided to discard the variable " area-per-worker ", the next step was to attempt to measure the rate at which risk of infection increased with compartment size. The notification rates were therefore correlated with workers-per-compartment to obtain estimating equations of the form

$$y=a+bW,$$

where y is the estimated notification rate, and W the number of workers in the unit.

Since the units were of widely different sizes, their notification rates were subject to different sampling errors. To allow for this a weighting system had to be introduced. The exact sampling distribution of the notification rates was not investigated, but it was assumed that they could be treated as binomials. The correct weight for each rate would then be

$$\frac{n}{p\,(1-p)},$$

where n is the number of workers on which the sample rate is based, and p the corresponding " true " proportion of workers notified. Values of p would normally have been estimated from the sample rates themselves, but the presence of many sample proportions of zero—implying infinite weight—made this impossible. The method adopted was to proceed in two stages. In the first stage all sample rates were given equal weight, and, from the equation thus calculated, weights were obtained for use in the second stage.

The second stage estimate of regression based on 807 compartment sizes and rates was

$$y=1\cdot40+0\cdot0096W.$$

According to this regression equation, the expected notification rate varies from 1·41 in the smallest unit encountered (one worker), to 4·40 in the largest (312 workers). That is to say, the extra hazards of factory employment can more than treble the risk of tuberculosis in the extreme case.

Is this the best estimating equation that can be obtained ? In Table IV are set out the actual distribution of notifications between units of various sizes, the distribution expected if risk of tuberculosis were independent of compartment size (Model " A "), and that expected on the basis of the equation above (Model " B "). It is clear that Model " B ", although a considerable improvement on Model " A ", is still far from being a satisfactory explanation of the way in which cases of tuberculosis were distributed. Compared with the expectation based on Model

TABLE IV

TEST OF MODELS "A", "B", AND "C"

Model "A": Risk independent of number of workers per compartment.
Model "B": Linear increase of risk with number of workers per compartment.
Model "C": Curvilinear increase of risk with number of workers per compartment (see Figure).

Compartment Size Group	Number of Workers in such Compartments, 1948	Number of Cases of Tuberculosis, 1940–49	Number of Cases Expected on Basis of Model "A", "B", or "C"		
			"A"	"B"	"C"
1 to 25	5,698	72	120·13	88·33	85·68
26 to 50	6,124	115	129·11	107·12	112·32
51 to 75	4,235	102	89·28	84·23	91·24
76 to 100	2,837	71	59·81	63·94	69·11
100 to 150	2,974	80	62·7	75·88	76·84
151 to 250	1,692	50	35·67	54·61	51·44
Over 250	868	25	18·3	36·37	28·72
Total	24,428	515	515·00	510·48	515·35
Degrees of Freedom left for χ^2 Test			6	5	4
Probability that Discrepancy between Expected and Actual Numbers of Cases could have arisen by Chance			$P<0·001$	$0·05>P>0·02$	$0·45>P>0·35$

"B", there is a deficiency of cases at each end of the range of compartment sizes and an excess in the middle. From a graph showing notification rates in these unit-size groups it appeared that a better fit to the data would be obtained by a curved line, possibly one tending to an upper limit. A systematic departure from linearity would justify a more complicated equation relating risk to numbers, but, before complicating the hypothesis, it is prudent to consider how else the appearance of curvilinearity could have arisen. It might occur, for instance, if either (*a*) the susceptible occupational groups were over-represented in the medium-sized compartments, or (*b*) the larger compartments contained groups of workers too remote to transmit infection to one another, causing the effective number of persons to fall below the total number present.

As far as (*a*) is concerned, it is impossible to disprove any such over-representation without becoming involved in a circular argument, since the "susceptibility" of an occupational group is inferred from its notification rate and this, in turn, is known to be largely affected by the size of the working units of which that occupational group is composed. But at least there is nothing amounting to evidence that occupational differences have caused an appearance of curvilinearity.

Possibility (*b*)—that large compartments may contain numbers of workers out of "infecting range" of each other—is that mentioned above in connexion with rough stuff workers. It has been rejected for a number of reasons. First, there is the evidence of an experimental demonstration by Williams (1950) of the rapidity with which exhaled droplets can reach all corners of a working unit. Secondly, droplets are not the only medium of infection to be considered: the notorious

hardihood of the tubercle bacillus enables it to survive in dust for long periods, and infected dust can persist in a room despite repeated and thorough sweepings (Lidwell, 1950; Lidwell and Lowbury, 1950). Thirdly, since operatives are not rooted in their place of work, " infecting range " may extend beyond the actual compartment.

(6) Second Measurement of Risk Gradient

It was therefore considered proper to try a non-linear form of estimating equation. There are precedents (deriving from Farr's " Law of Density ") for describing cognate material in terms of an equation of the form

$$y = a\,x^n,$$

where y is a mortality or morbidity rate, x an independent variable, and n a positive constant smaller than unity (Brownlee, 1920; Cheeseman and others, 1939). There is no doubt that to use a curve of this type here would result in a better fit to the data, as would also the addition of a term $-cW^2$ to the equation already used. But both these types of curves have the disadvantage that they do not tend to an upper limit, and that they cannot be interpreted in terms of any known mechanism of infection. Instead of using them, therefore, an exponential form has been adopted, of which the rationale is as follows:*

Suppose that in the course of daily life a person is in touch with F " effective contacts "—effective in the sense that, if any *one or more* of them is a carrier of a certain disease, then he or she is in contact with the disease. (The number F will not, of course, be the same for, say, airborne as for contagious diseases.) The total of " effective contacts " may be thought of in two parts, of which U represents the contacts made at work, and D all other contacts. For large groups of workers it is reasonable to treat D as a constant, and U as a variable, whose value depends upon the specification of the particular group. In the present example, U will be set to equal the number of persons assumed to be contacted at work, i.e. one less than the total number of workers in the same compartment. Now let π be the proportion of carriers in a community and R the risk of making contact with the infection. It follows that

$$R = 1-(1-\pi)^{D+U} \qquad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (i)$$

We may also suppose that $R = y/M$, where y, as before, is the expected notification rate and M the percentage of the community which, once in " effective contact " with tuberculosis, will develop the disease within a specified period. As the number of " effective contacts " increases, the risk R approaches unity, and y approaches its upper limit M. Substituting y/M for R in Equation (i) and expressing it in the more convenient logarithmic form we have

$$\log\,(1-y/M) = D\,\log\,(1-\pi)+U\,\log\,(1-\pi) \qquad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (ii)$$

The percentage of any group observed to develop tuberculosis obviously depends in part on the length of time for which the group is under observation. By expressing each notification rate as a fraction of the ten-year maximum rate, we can make an

* Since writing this paper we have found the same argument applied by Hogben (1947) to the risks of jaundice following transfusion with pooled plasma or serum.

adequate allowance for this time factor. But there is a more subtle way in which the passage of time complicates the assessment of risk. A person continuously in contact with F others will not always be in contact with the same F individuals. Therefore in a long period he will have a total number of contacts which exceeds F. No correction has been attempted for this second factor and, as a consequence, the estimates of π and D are each subject to an upward bias.

The compartments were arranged in twelve size groups. The notification rate y was calculated for each group and also a weighted average value of U for all units in the group. The weighted average used was $\Sigma W(W-1)/\Sigma W$. The twelve values of y were plotted against values of U and a freehand curve drawn to obtain an estimate of 3·50 per cent. for the upper limit M. Each value of y was divided by M to express the risk of infection in the unit size group. The least-squares method, with appropriate weighting, was used to determine the risk equation in its logarithmic form. It was then expressed in exponential form and the associated correlation coefficient found to be /0·215/ based on 804 degrees of freedom, compared with +0·16 based on 805 degrees of freedom for the linear equation above.

The equation finally obtained in this way was

$$y=3\cdot50\ \{1-(1-0\cdot0085)^{51+U}\} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (iii)$$

That is to say, the carrier rate was estimated at 0·85 per cent., and the number of effective contacts from domestic and other non-working sources as 51. When U is set equal to zero, y has the value 1·23 compared with an average notification rate of 2·11. This implies that more than 40 per cent. of the cases of tuberculosis occurring in industry as a whole were the result of infection at work. In the larger compartments the proportion of cases so caused is, of course, higher, rising to about 60 per cent. in the largest units of all. The distribution of cases expected on the basis of the new equation is shown in Table IV (Model " C "), which illustrates the progress made by adopting a non-linear estimate of risk. In order to maintain the earlier Model " B " it would have been necessary to accept that a chance of about 1 in 25 had occurred. But if Model " C " is correct, then the differences between actual and expected cases are no larger than would occur in more than 1 in 3 of repeated samples.

(7) Effect of Factory Size

Even Model " C " does not exhaust the possibilities of accounting for variations in the notification rates in terms of contacts made at work. In Table V (overleaf) the actual notification rates in a number of factory size groups are compared with the rates which would have occurred if cross-infection within the factory had been confined to infection within compartments. It is evident that over and above the increase of risk with compartment size there is a further increase associated with factory size.

For each room in a factory the number of cases or fractions of case expected on the basis of Model " C " were summed, and the factory totals were summed in ten factory-size groups. A χ^2 test confirmed that the departure of actual from expected cases was statistically significant.

The close simultaneous relation between compartment size and factory size made

TABLE V
RESIDUAL FACTORY SIZE EFFECT ON RISK

Factory Size Group	Expected Notification Rate based on Model " C "	Actual Notification Rate	Difference
1– 50	1·54	0·98	−0·56
51– 75	1·50	1·64	+0·14
76–100	1·70	1·20	−0·50
101–150	1·87	1·25	−0·56
151–200	1·96	1·37	−0·59
201–250	2·02	1·82	−0·20
251–300	2·38	2·95	+0·57
301–500	2·27	2·46	+0·19
501–600	2·41	3·42	+1·01
Over 600	2·72	3·14	+0·42

it impracticable to include them both as " independent " variables in the same estimating equation. Otherwise we could have obtained a still better estimator of risk of the form $y=M\{1-(1-\pi)^{D+U+Q(T-U)}\}$, in which T is the total number of workers in the factory.

One result of taking factory size into account would have been a substantial increase in the estimated proportion of cases resulting from infection at work. Another result would have been to reduce the estimate of D.

DISCUSSION

Is 40 per cent. an unreasonably high estimate of the proportion of tuberculosis cases originating in infection at work ? A preoccupation with the contacts who can most easily be identified (i.e. those in the home and close friends) has given rise to the impression that tuberculosis is only passed between persons whose relationship is more intimate than that of fellow workers. But since it is virtually impossible to develop human type tuberculosis without meeting an infectious person, the figure 40 per cent. is actually quite small. Only 1 in 4 of Northamptonshire boot and shoe factory operatives who developed tuberculosis between 1920 and 1948 had any history of family contact, and among 2,829 pulmonary cases notified in Glasgow during 1949 only 509 (18 per cent.) had *ever* lived in the same house with a tuberculosis case of any kind (Laidlaw, 1949). In all, therefore, about three-quarters of tuberculosis cases remain to be accounted for in terms of non-household contacts. Since this is so, the remarkably good fit obtained with the second form of estimating equation justifies a discussion of the theoretical model on which it is based.

To illustrate the discussion, a diagram has been drawn in which risk of infection R is shown as a function of number of effective contacts F, on four different assumptions about the carrier rate π. These are the curves G, H, J, and K. For G the value of π is set equal to the percentage of newly-discovered active cases of tuberculosis found by mass radiography (1945-46) amongst Northamptonshire workers *not* in the boot and shoe trade (i.e. 0·33 per cent.); for H to the corresponding percentage amongst boot and shoe workers (i.e. 0·59 per cent.); for J the value

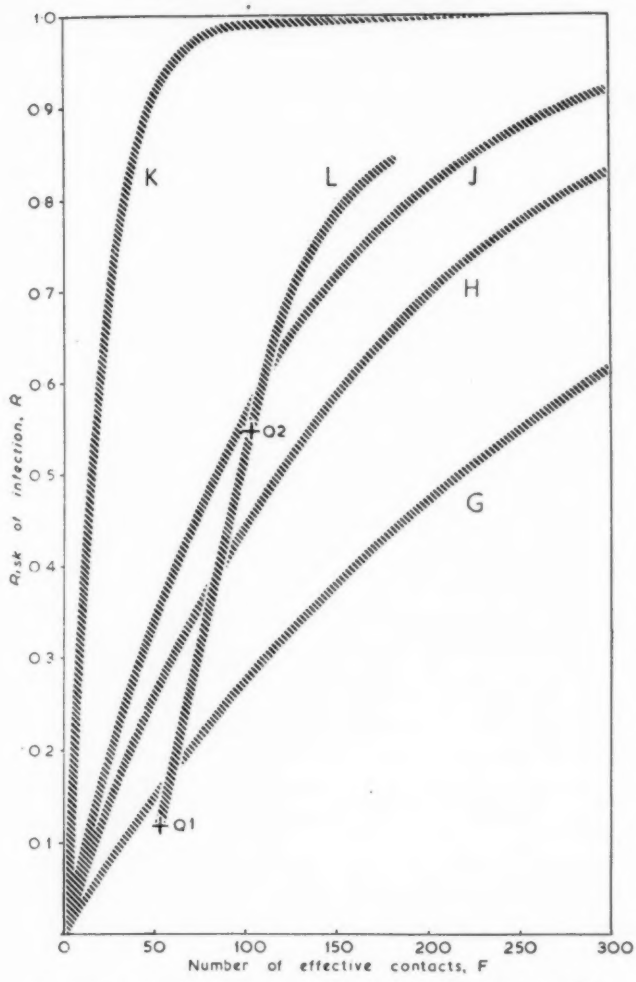

FIGURE.—Graph to illustrate discussion of Model " C ".

of π is that estimated from notification data in the present analysis (i.e. 0·85 per cent.); for K the value of π has been set at the arbitrary figure of 5 per cent.

The Figure shows clearly the special relevance of the theoretical model to diseases with low carrier rates. Where there are large numbers of carriers (5 per cent. is a very modest estimate of the carrier rate for the common cold at certain times of the year) then only a few contacts are needed to subject a person to his maximum risk of infection. But when the carrier rate is below 1 per cent., meeting an extra one or two hundred persons in the course of the day's work can easily double or treble the risk of infection.

According to the diagram we should expect the incidence of tuberculosis to rise whenever two or more groups of persons are thrown together for the first time. This phenomenon is well known when civilized and primitive societies meet, but is one which the medical profession is apt to overlook when attempting to explain the prompt rise in the incidence of tuberculosis (and also of infective hepatitis) on the outbreak of war.*

The same argument applied to individuals throws light on morbidity at two

* During the first half of the 19th century there was an unprecedented migration of country folk into towns accompanied by explosive outbreaks of many infectious diseases. Since real wages were increasing and food supplies improving during the relevant period (Clapham, 1926), it is impossible to ascribe the outbreaks to a decline in the standard of living. One has, therefore, to conclude that the sudden collision of large numbers of susceptible persons was alone sufficient to cause giant epidemics of diseases such as cholera, typhoid, scarlet fever, and diphtheria, which in their endemic phase have low carrier rates. In this connexion it is of interest to note that during the long spell of unemployment in the cotton industry brought about by the American Civil War, the death rate in several Lancashire towns temporarily declined (Simon, 1862). Towards the end of the century when boot and shoe factories began to replace the old cottage workshops there was undoubtedly an improvement in living and working conditions, yet the excess of tuberculosis in the boot and shoe industry dates from this period (Cairns and Stewart, 1951).

particular ages. The first is that of the child moving from a home to a school environment at the age of five. At this age the peak incidence of measles and of whooping cough has already been passed, but the rarer exanthemata, such as scarlet fever, occur more frequently than before. The second is that of young adults, moving from a school to a working community, when a rise in the incidence of tuberculosis and lobar pneumonia occurs. So long as these two diseases are thought of solely in terms of resistance it is difficult to explain this phenomenon, but if they are once thought of in terms of infection the paradox is resolved. In the case of tuberculosis, the incidence of the disease increases even more than would be expected from the curves in the diagram. This is probably due to the fact that the school-leaver, besides increasing his number of effective contacts, is moving into an adult community in which the carrier rate is relatively high. That is to say, besides moving to the right along one of the risk curves, he also shifts onto a higher curve.*

One of the assumptions which had to be made in the present calculations was that carriers were distributed at random. This may have been true originally in the boot and shoe trade, and may still be true in more strenuous trades, where the nature of the work tends to eliminate new cases of tuberculosis as they arise. But in comparatively light work, such as shoemaking, provided no deliberate measures are taken to eliminate cases, there will always be an excess of carriers in those parts of the industry where the most infection has been taking place, that is, in the largest working units. These are precisely the places in which the most chronic cases have been found and where, man for man, they produce the most victims (Stewart and Hughes, 1951). It follows that any given percentage of carriers in the boot and shoe industry as a whole will do more damage than the same percentage in an industry where carriers are evenly distributed.

The non-random distribution of carriers will have given an upward bias to the *ex post* estimate of the carrier rate (in addition to the bias already mentioned). Without this bias curve *J* would lie nearer to curve *H*. Curve *L* illustrates what would happen if there were *no* elimination of new cases: an entrant to the industry who added only fifty to his number of effective contacts would move from the point Q^1 to Q^2, a fivefold increase of risk.

Multiplication of risk on this scale wholly outweighs any effect due to age, sex, occupation, spacing of workers, and differences between " good " and " bad " factories as judged by factory inspectors. The first three of those variables are in any case not amenable to control, and neither re-spacing workers nor reconditioning factories, however desirable, is likely to have an appreciable effect on tuberculosis morbidity. There remain, therefore, only two courses open: to reduce the carrier rate, or to reduce the number of " effective contacts ".

* The "control" group of young persons used in the Prophit Survey (Daniels and others, 1948) were said to be " at no greater risk of encountering infection than any town-dweller ". Of controls under 17 years old, 76 per cent. were tuberculin positive, and of those between 18 and 20, 81 per cent. But in the course of B.C.G. trials in the industrial Midlands it has been found that out of 2,700 children in their last year at school only 969 (36 per cent.) were found to be positive (Hughes, 1951). If these findings are to be reconciled, we must assume that the amount of primary infection taking place in the first years after leaving school is far greater than in any other period of life.

The first of these courses is the more promising. In mass radiography there exists an ideal weapon for the detection and removal of sources of infection. Active cases of tuberculosis discovered amongst male shoemakers in the 1945-46 survey were, on the average, 8 years older than the male cases notified other than by mass radiography during the same period.* This illustrates how such surveys can locate the chronic male cases which otherwise tend to escape diagnosis, and which constitute the hard core of infection. It is too early to assess how far this first survey has succeeded in reducing the rate of current infection, but if the theoretical model is correct, an initial halving of the carrier rate should have reduced the average risk of infection by more than half. In this connexion it is of interest to note that in the repeat mass radiography survey of Northamptonshire (1948-49) active tuberculosis was found no more frequently among shoemakers than among other workers, *even in those individuals* x-*rayed for the first time* (Fisher, 1951).

The pattern of infection revealed by recent research shows how to make continued control by mass radiography at once more efficient and more economical. The correct strategy will be to concentrate on the largest factories, where sources of infection are most frequent, most dangerous, and, for administrative reasons, easiest and cheapest to find. If, however, case-finding by mass radiography is pursued without providing alternative employment for tuberculous subjects after treatment, it may result in a falling off of volunteers in just those sections of the working population which it is most important to examine. This difficulty could be avoided if special factories, similar to those already provided under the Ministry of Labour Remploy Scheme, were available for all tuberculous subjects who are fit for work. Even though known tuberculous subjects are less dangerous sources of infection than untreated cases, it is important from the point of view of policy not to discrimate between these two types of case. For if it became generally known that suitable working conditions and economic security were guaranteed for *all* tuberculous persons, reluctance to submit to periodic examination might be reduced to vanishing point. At present a great deal of attention is paid to protecting members of infected households, but persons equally in need of protection are fellow-workers from uninfected houses.

The second possible line of action, namely reduction of the number of effective contacts, would in practice be difficult, if not impossible. The present tendency in the boot and shoe industry, as in others, is for factories to grow larger. This tendency is accelerated by the modern preference for vast, multi-process workshops instead of separate rooms for each process. More partitions, or at least the preservation of existing partitions, would be of some protective value against tuberculosis, and perhaps also against that troublesome cause of absenteeism, the common cold.

SUMMARY

At least 40 per cent. of the cases of tuberculosis occurring amongst workers in

* The average ages of these groups were (numbers of cases in brackets): mass radiography notifications, 45·3 years (56); other notifications, 37·5 (66). A similar age relationship was found for male workers in other trades.

Northamptonshire boot and shoe factories during 1940-49 may be attributed to infection at work. For large factories the figure may be as high as 60 per cent.

There is a definite association between risk of tuberculosis and numbers of persons working together, but no perceptible association between risk of tuberculosis and " overcrowding ".

The hypothesis that risk of tuberculosis varies in direct proportion to the number of contacts at work, though not entirely worthless, is shown to be unsatisfactory. A second hypothesis is developed according to which risk bears a curvilinear relation to number of contacts. This provides an adequate explanation of the observed facts. From this hypothesis it is argued that:

(1) Any given percentage of active cases of tuberculosis in a light industry constitutes a greater danger to fellow-workers than the same percentage in a heavy industry.
(2) An excess of tuberculosis amongst boot and shoe workers implies neither sub-average powers of resistance, nor the existence of a specific occupational hazard, nor bad working or living conditions.
(3) There is nothing paradoxical in the rising incidence of tuberculosis at young adult ages, although acquired specific resistance is higher at this age than in childhood.

Mass radiography has a vital role to play in the prevention of tuberculosis. Suggestions are made for the most efficient use of mass radiography resources.

The hypothesis advanced may have relevance outside the industrial field, and may apply to communicable diseases other than tuberculosis.

Our thanks are due to members of the boot and shoe industry who allowed us access to their factories, and to the Medical Officers of Health for Northampton County Borough and for Northamptonshire who put their records at our disposal. In the writing of the paper we have benefited from the advice of our colleagues, Dr. W. T. Russell and Dr. I. Sutherland, and of Mr. P. A. P. Moran of the Oxford University Institute of Statistics. The Medical Research Council contributed generously towards the expenses of the investigation.

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ABO GROUPS OF INFANTS AND CHILDREN DYING IN THE WEST OF SCOTLAND (1949-1951)

BY

DAVID STRUTHERS

Department of Pathology, Royal Hospital for Sick Children, Glasgow

Hirszfeld and Zborowski (1925) and Levine (1943) found fewer group A children in AO families (father group A, mother group O) than in OA families (father group O, mother group A). The statistical significance of this deficiency of group A children was firmly established by Waterhouse and Hogben (1947) who reckoned that 25 per cent. of the expected number of group A children were missing from the AO families of the general population. By Bernstein's hypothesis, however, group A children are conceived with equal frequency in AO and in OA families, and it therefore follows that the missing children must have died some time between conception and childhood.

Apart from ABO iso-immunization, or some similar mechanism involving the natural alpha-antibodies carried by the mothers of AO families, all factors responsible for the loss of group A children should operate alike in AO and OA families. Thus Waterhouse and Hogben concluded that ABO iso-immunization was probably responsible for the deficiency of group A children observed in AO families (i.e. for the loss of about 3 per cent. of all conceptions in the general population), the total loss of life from haemolytic disease of the newborn due to Rh-sensitization being less than 0·5 per cent. of all conceptions.

The fate of the missing children has yet to be ascertained. Because ABO iso-immunization does not contribute materially to the incidence of haemolytic disease, and because the agglutinins concerned are normally present in the maternal blood early in pregnancy, Waterhouse and Hogben suggested that ABO iso-immunization acts early in foetal life, causing abortions and miscarriages. It is undoubtedly true that very few of the missing children die of haemolytic disease, but it may be argued that immaturity of the agglutinogens makes it impossible for ABO iso-immunization to act early in foetal life. Furthermore, the absence of a significant deficiency of group A among the newborn of group O mothers in a series of 2,000 births reported by Boorman (1950), and in a series of 7,856 births reported by Bryce and others (1950), suggests that some of the observed deficiency of group A children may have arisen after rather than before birth, an excess of group A children born of group O mothers having died at an early age from conditions other than haemolytic disease.

Present Investigation

The ABO groups of a random series of infants and children dying before the age of 13 years are compared in this work with those of the adult population to which their parents belong.

The null hypothesis here assumes that all the missing victims of ABO iso-immunization die before birth, and, in this event, the frequency of group O should be similar among the dying offspring and among their parents. It is true that ABO iso-immunization may have left an excess of group O, relative to the frequency of the corresponding gene, in the population to which the parents belong, but any deficiency of group O from this cause, among the offspring conceived, will be more than offset by the assumed loss of heterospecific foetuses.

Except for the remote possibility that some hitherto unsuspected mechanism may be responsible for an appreciable mortality after birth among homospecific as well as heterospecific infants of groups A, B, and AB, the foregoing null hypothesis would be disproved by finding a significant deficiency of group O among the infants and children dying after birth. It would, then, follow that some, at least, of the missing victims of ABO iso-immunization must die after birth.

Material and Methods

Post-mortem samples of blood were collected from 400 consecutive infants and children examined in this department between March, 1949, and March, 1951. In each case the ABO group was determined by the " open slide " technique, and in sixty consecutive cases the direct anti-human globulin test was applied as described by Mollison and others (1948). Apart from infectious diseases such as measles and whooping cough which generally account for very few deaths under the age of one year, most of the common causes of death under the age of 13 years in the West of Scotland are here represented.

The diagnosis of broncho-pneumonia was confirmed histologically in 148 cases in the series and includes all grades ranging from frank pulmonary suppuration to a massive haemorrhagic consolidation with minimal infiltration by polymorphonuclear cells.

In a series of 6,011 donors examined during 1948, the Glasgow and West of Scotland Blood Transfusion Service found:

Group	Per cent.
O	50·96
A	35·18
B	10·87
AB	2·98

Results

The distribution of ABO groups in the present series of infants and children arranged according to their ages at death is presented in Table I. Compared with the adult population to which the parents belong, there is a highly significant deficiency of group O in the series, particularly among the infants dying between the ages of 4 weeks and 1 year.

Within the series there is also a highly significant difference in the frequency of group O in cases with and without autopsy evidence of broncho-pneumonia (Table II). In 55 cases with no apparent abnormality other than broncho-pneumonia at autopsy the frequency of group O is as low as 25·5 per cent. as against 50·0 per

TABLE I

NUMBERS AUTOPSIED AS A PERCENTAGE OF THE TOTAL BY AGE GROUPS

Age at Death	ABO Group				Totals	Deficiency of Group O* ±Standard Error
	O	A	B	AB		
Under 4 weeks	54	41	18	9	122	6·70±4·53 per cent.
	(44·3)	(33·6)	(14·7)	(7·4)		t=1·48
						P=0·2
4 weeks to 1 year	62	71	17	8	158	11·72±3·94 per cent.
	(39·2)	(44·9)	(10·8)	(5·1)		t=2·97
						P<0·01
Total 0–1 year	116	112	35	17	280	9·53±3·01 per cent.
	(41·4)	(40·0)	(12·5)	(6·1)		t=3·16
						P<0·01
1–2 years	16	17	7	—	40	
Total 0–2 years	132	129	42	17	320	9·71±2·83 per cent.
	(41·3)	(40·3)	(13·1)	(5·3)		t=3·43
						P<0·001
2–13 years	41	29	8	2	80	−0·29±5·59 per cent.
	(51·2)	(36·3)	(10·0)	(2·5)		t=0·05
						P>0·90
Total 0–13 years	173	158	50	19	400	7·71±2·56 per cent.
	(43·3)	(39·5)	(12·5)	(4·7)		t=3·01
						P<0·01

* Expected frequency of Group O=50·96±0·65 per cent. as quoted for the adult population of the West of Scotland, 1948.

TABLE II

NUMBERS AUTOPSIED

ABO Groups	Broncho-pneumonia			
	Present		Absent	
	No.	%	No.	%
O	47	31·8	126	50·0
	Difference=18·24±4·95 per cent.; t=3·68; P<0·001			
A	68	45·9	90	35·7
B	21	14·2	29	11·5
AB	12	8·1	7	2·8
Totals	148	100	252	100
χ^{2}*	28·17		0·21	
$P(N=3)$	<0·001		>0·9	

* Comparing each of the above distributions with that of the 6,011 donors quoted.

cent. for the cases with no broncho-pneumonia (difference 24·5±6·7 per cent.; t=3·7; P<0·001). Furthermore, by the χ^2 test, the chances of obtaining the individual distributions set out in Table II from a population with the ABO groups distributed as in the adult population of the West of Scotland are respectively less

than 1 in 1,000 for cases with broncho-pneumonia and more than 900 in 1,000 for those without autopsy evidence of broncho-pneumonia. Frank pulmonary suppuration was present in 52 cases, of which 30·8 per cent. belong to group O, the corresponding figure in 81 cases of haemorrhagic broncho-pneumonia being 28·4 per cent. This difference (2·4±8·1 per cent.; $t=0·3$; $P<0·9$) is insignificant.

The higher frequency of group O among neo-natal deaths than among deaths between the ages of 4 weeks and 1 year in the series appears to result from a relative excess of group O infants dying from conditions peculiar to the neo-natal period, i.e. from haemolytic disease of the newborn, intracranial haemorrhage, vernix pneumonia, haemorrhagic disease, and from congenital malformations inconsistent with an independent existence. In 54 such cases the frequency of group O is 57·4 per cent., as against 33·2 per cent. in the 68 neo-natal deaths attributable to broncho-pneumonia, infantile diarrhoea, and other conditions commonly responsible for the death of infants over the age of 4 weeks. This difference is significant (24·2±8·9 per cent.; $t=2·7$; $P<0·01$). Haemolytic disease of the newborn and congenital malformations account here for most of the apparent excess of group O infants dying from conditions peculiar to the neo-natal period.

Rh antibodies were found in the maternal blood in ten out of thirteen cases of haemolytic disease in the series (group O 10, group A 3). In one remaining case the infant was group A, Rh-positive, and the mother was group O, Rh-positive.

Routine histological examination revealed twelve cases of pancreatic fibrosis in the series, broncho-pneumonia being present in ten of the cases concerned. One of the group O infants affected suffered from haemolytic disease, Rh antibodies being detected in the maternal blood. The groups of the other nine infants with pancreatic fibrosis were as follows:

group O	2
group A	5
group B	2
group AB	2

Apart from known cases of haemolytic disease, the anti-human globulin tests were negative in the sixty cases examined.

Applying the distributions of the ABO groups among the parents to the observed numbers of group O in the series, the total excess of group A, B, and AB deaths corresponds here to roughly 15 per cent. of the series. Among the infants examined the corresponding figure is about 19 per cent.

Conclusions and Discussion

It may be concluded from the above analysis that an association probably existed here between the antigens A and B and infant death with autopsy evidence of broncho-pneumonia.

If there is, however, an appreciable deficiency of group A children in the AO families of the West of Scotland to-day and if this deficiency is not apparent among the newborn, it follows that the mechanism of ABO iso-immunization described

by Waterhouse and Hogben in the general population must have contributed to the selective mortality observed herein among infants of groups A, B, and AB dying from broncho-pneumonia or from conditions complicated by broncho-pneumonia.

The infant mortality rate for the West of Scotland, 1949-51, is probably 40-50 per 1,000 live births, and it is apparent that the observed excess of group A deaths cannot account for the loss of 25 group A infants per 1,000 conceptions, i.e. for 25 per cent. of the group A children expected in the AO families of the West of Scotland. Conditions excluded from the present series may be responsible for a further selective loss of group A children, but it should be observed that the 25 per cent. deficiency refers to families examined between 1927 and 1945 and hence to a population with an infant mortality rate which may have been very much higher than 50 per 1,000 live births.

Smith (1945) found a specific maternal antibody response in forty out of 46 normal heterospecific pregnancies, and it is generally agreed that the alpha-beta antibodies of the newborn are maternal in origin. There is, therefore, good reason to believe that ABO iso-immunization may operate to some extent in many normal heterospecific pregnancies, although it rarely causes haemolytic disease of the newborn and may even protect the Rh-incompatible, heterospecific offspring from haemolytic disease due to Rh-sensitization.

The negative anti-human globulin tests above may indicate that the maternal alpha-beta antibodies were taken up by extra-cellular group specific receptors rather than by the foetal red cells. Alternatively, the test concerned may be specific for the antibodies in haemolytic states rather than for sensitizing antibodies in general. This alternative does not necessarily imply that the " anti-human globulin reacting " antibodies cause the associated haemolytic reaction. The destruction, by haemolysis, of red cells sensitized with some primary antibody substance may lead to the liberation of modified antibodies reacting with the anti-human globulin serum on readsorption onto circulating red cells.

Obviously many more cases must be examined to establish an association between pancreatic fibrosis and the AB antigens. There seems to be no reason, however, for distinguishing here between the broncho-pneumonia in such cases and the general group of broncho-pneumonia cases to which the foregoing conclusions refer. Glanzmann (1946) and Eggimann (1949) have already suggested that Rh antibodies may be responsible for some cases of fibrocystic disease of the pancreas, and it is therefore conceivable that maternal-foetal incompatibility in respect of the ABO, Rh, and other antigen systems may contribute to the complex genetic pattern of infant deaths from broncho-pneumonia associated with fibro-cystic disease of the pancreas.

Finally, by way of illustration, two of the possible mechanisms are outlined whereby the maternal alpha-beta antibodies may contribute to infant death from broncho-pneumonia.

(1) The maternal antibodies may diminish the resistance of heterospecific offspring to certain infections. In fibro-cystic disease of the pancreas it is thought that pancreatic

dysfunction leads to a deficiency of vitamin A which, in turn, predisposes to broncho-pneumonia. The normal pancreas is known to be unusually rich in the specific AB substances and it may be that, short of actual fibrosis, a local accumulation of AB antigen-antibody complexes interferes with the normal mechanism of pancreatic function.

(2) The foetal red cells may be sensitized by antibodies which do not react with the anti-human globulin serum and may even tend to inhibit haemolytic activity, e.g. by contributing to the local balance of cholesterol compounds rather than to that of lyso-lecithins. Such antibodies could be responsible for a massive intravascular conglutination or agglutination reaction within the pulmonary circulation, possibly precipitated by, or else predisposing to, certain local infections. Haemorrhagic pulmonary lesions are frequently present in fatal cases of icterus gravis and it seems reasonable to suggest that the extent of any such local conglutination reaction will vary inversely with the rate at which the affected cells undergo haemolysis.

Summary

According to authorities quoted, ABO iso-immunization is responsible for the loss of 25 per cent. of the group A children expected in AO families (father group A, mother group O), i.e. for the loss of 3 per cent. of all conceptions in the general population. It has been suggested that the children concerned die before birth. Recent surveys of the newborn and of their mothers have not, however, revealed a significant deficiency of group A among the newborn of group O mothers. It is suggested here that, in this event, many of the missing children must die after birth from conditions other than haemolytic disease.

In this work the ABO groups of a consecutive series of 400 infants and children autopsied in this department are compared with those of the adult population to which the parents belong. A highly significant deficiency of group O (i.e. an excess of groups A, B, and AB) was found in the series, particularly among the infants with autopsy evidence of broncho-pneumonia.

It was concluded that an association probably existed here between the A and B antigens and infant death from broncho-pneumonia and from conditions complicated by broncho-pneumonia. If there is, however, a deficiency of group A children in the AO families of the West of Scotland to-day, and if this deficiency is not apparent among the newborn, it follows that the mechanism of ABO iso-immunization described in the general population probably contributed to the foregoing association.

I am indebted to Dr. J. Wallace and to Mr. G. R. Milne of the Glasgow and West of Scotland Blood Transfusion Service for the necessary anti-AB and anti-human globulin sera and for the distribution of ABO groups in the adult population of the West of Scotland.

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SEX RATIO* OF STILLBIRTHS RELATED TO BIRTH WEIGHT

BY

THOMAS McKEOWN and C. R. LOWE

From the Department of Social Medicine, University of Birmingham

It is well known that more males than females are stillborn: of stillbirths notified in England and Wales (1939-47), 53·8 per cent. were males. This is because:

(1) There are more male foetuses at risk, for, as indicated by the sex ratio of stillbirths and live births combined (51·5 for England and Wales, 1939-47), there are more males than females in the uterus at 28 weeks, the time from which stillbirths are notified. The male excess at 28 weeks must result from one or both of two causes:

(*a*) a high sex ratio at conception
(*b*) a greater loss of females than of males as abortions in the period between conception and notification.

On the first point there is no direct evidence, and on the second it is generally believed that the sex ratio of abortions is above 50. But reasons have been suggested in support of the view that the gross inadequacy of data from the early months of gestation must leave the sex ratio of abortions, and hence the sex ratio at conception, an open question (McKeown and Lowe, 1951).

(2) Stillbirth rates are higher for male than for female births; this follows from the fact that the sex ratio of stillbirths is higher than that of total births. In England and Wales (1939-47), the sex-specific stillbirth rates (per thousand related total births) were 31·8 and 29·0 for males and females respectively. Various explanations have been suggested for this difference, but none is supported by any substantial evidence.

The purpose of this communication is to explore the association between the sex ratio of stillbirths and birth weight. It is known that weight distributions are different for male and female births, and the incidence of certain causes of death (for example difficult labour) is associated with birth weight and is different for the two sexes. Hence it is of interest to enquire whether the difference in the sex-specific stillbirth rates is reduced when births of corresponding weight are compared. If it is found to be reduced, this would in part account for the fact that the sex ratio of stillbirths is higher than that of the total births from which they are drawn.

* Sex ratio is expressed as the percentage of males $\frac{M}{M+F} \times 100$.

Material and Results

For the years 1945-49 we obtained from city records data for all stillbirths notified in Birmingham. During this period the Maternity and Child Welfare Department, assisted by the attendant at the birth (doctor or midwife), completed a card in respect of each stillbirth. The information recorded included sex, birth weight (lb. and oz.), and the certified cause of stillbirth. In the 5 years there were 2,388 stillbirths, for seventeen of which birth weight was unspecified. The requisite data are thus available for 2,371 stillbirths.

We also make use of valuable data recently published by Baumgartner and others (1950), which give the sex and birth weight (g.), but not the cause of death, for foetal deaths subsequent to 20 weeks of gestation notified in New York City during the two years 1939-40. There were 6,978 foetal deaths, for 1,871 (27 per cent.) of which weight was unspecified. The authors have assigned weights to these 1,871 births according to the duration of gestation, using information about the correlation between gestation and weight obtained from the stillbirths for which both variables were recorded.

Weight distributions of the related populations of live births are available for the New York data, and for Birmingham data for one year only (1947).

TABLE I

Sex Ratio of Births subsequent to 28 weeks of Gestation related to Birth Weight (Birmingham)

Birth Weight (lb.)	Stillbirths (1945–1948)			Total Births (1947)		
	Percentage Distribution		Sex Ratio	Percentage Distribution		Sex Ratio
	Males	Females		Males	Females	
Under 3	10·1 (127)	15·8 (176)	41·9	0·8 (92)	0·7 (72)	56·1
3–	9·7 (122)	11·3 (126)	49·2	0·7 (87)	0·8 (82)	51·5
4–	11·8 (149)	12·8 (143)	51·0	1·7 (195)	2·0 (225)	46·4
5–	11·5 (145)	12·8 (142)	50·5	6·0 (701)	8·1 (895)	43·9
6–	16·4 (206)	16·8 (187)	52·4	20·4 (2,402)	25·7 (2,825)	46·0
7–	19·0 (239)	18·1 (201)	54·3	34·8 (4,096)	36·1 (3,969)	50·8
8–	12·8 (161)	8·5 (95)	62·9	24·5 (2,880)	20·0 (2,202)	56·7
9–	5·4 (68)	2·7 (30)	69·4	8·5 (1,005)	5·2 (572)	63·7
10 and over	3·3 (41)	1·2 (13)	75·9	2·7 (317)	1·3 (147)	68·3
Total ..	100 (1,258)	100 (1,113)	53·1	100 (11,775)	100 (10,989)	51·7

The data can be examined in various ways, of which perhaps the most satisfactory is to begin by noting that:

(*a*) the sex ratios of stillbirths (all weights) are 53·1 and 55·0 for Birmingham and New York respectively
(*b*) the sex ratios vary with birth weight (Tables I and II).

In Birmingham the ratios increase fairly regularly with increasing weight; in New York the same is true, with the difference that the sex ratio is distinctly raised for births at the lowest weight recorded (" under 1,000 g.": sex ratio 58).

We must first consider to what extent the association between the sex ratio and the weight of stillbirths is determined by the relation between the sex ratio and the weight of total births. Table I shows that the sex ratio of total births (Birmingham) decreases up to 5 lb., and then increases regularly in each weight group. There is

TABLE II

SEX RATIO OF BIRTHS SUBSEQUENT TO 20 WEEKS OF GESTATION RELATED TO BIRTH WEIGHT (NEW YORK CITY 1939–1940)

Birth Weight (g.)	Stillbirths			Total Births		
	Percentage Distribution		Sex Ratio	Percentage Distribution		Sex Ratio
	Males	Females		Males	Females	
Under 1,000	28·8 (1,104)	25·4 (799)	58·0	1·5 (1,662)	1·2 (1,289)	56·3
1,000–	9·9 (381)	12·0 (378)	50·2	1·0 (1,071)	1·0 (1,079)	49·8
1,500–	9·0 (344)	9·7 (306)	52·9	1·6 (1,741)	1·8 (1,903)	47·8
2,000–	9·5 (366)	9·7 (305)	54·5	4·7 (5,203)	5·8 (6,072)	46·1
2,500–	9·5 (364)	10·1 (316)	53·5	16·1 (17,909)	21·9 (22,916)	43·9
3,000–	12·9 (496)	15·7 (493)	50·2	36·9 (41,199)	40·0 (41,770)	49·7
3,500–	10·8 (415)	9·9 (311)	57·2	27·5 (30,687)	21·9 (22,864)	57·3
4,000–	5·3 (202)	4·7 (149)	57·5	8·8 (9,811)	5·4 (5,618)	63·6
4,500–	2·8 (109)	1·9 (59)	64·9	1·7 (1,926)	0·8 (871)	68·9
5,000 and over	1·5 (57)	0·8 (24)	70·4	0·3 (309)	0·1 (133)	69·9
Total ..	100 (3,838)	100 (3,140)	55·0	100 (111,518)	100 (104,515)	51·6

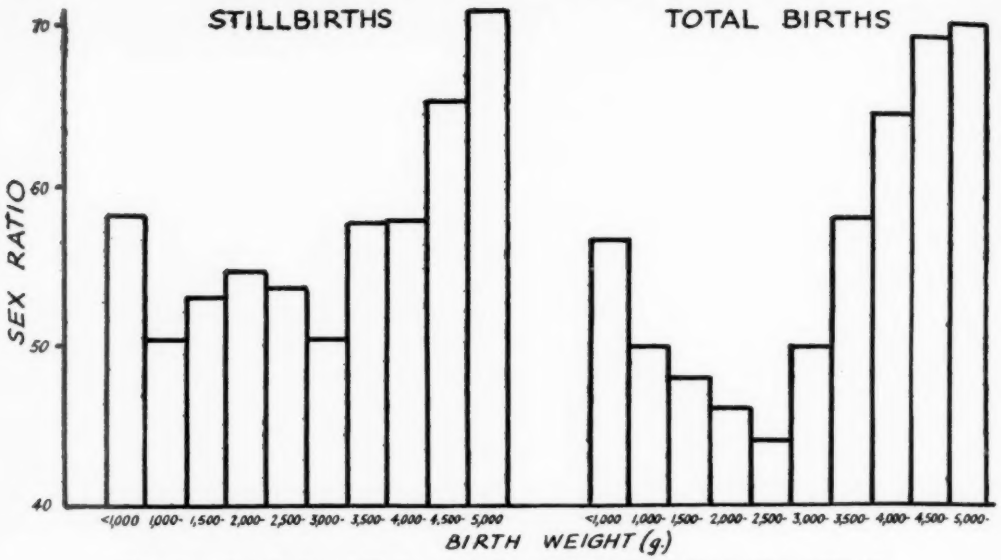

FIGURE.—Sex Ratio of Births related to Weight, New York City, 1939-40

an excess of males at the lowest weights, and a marked excess at the highest weights, but in three weight groups (4, 5, and 6 lb.) there are more females than males. New York data exhibited in Table II (see also Figure) are entirely consistent with these observations, although no strict comparison can be made because of the different weight systems in which the observations are recorded. It is evident that the association between the sex ratio and the weight of stillbirths in part reflects the weight and sex composition of the population of total births from which they are drawn.

The second question to be considered is the relation between sex-specific stillbirth rates and birth weight, for any difference between sex ratios of total births and stillbirths in each weight group reflects the differential mortality of males and females. For this purpose we require weight distributions of the related populations of total births, and, as indicated above, this is available in Birmingham for 1947 only. Tables III and IV show what is well known, namely that stillbirth rates are high at low weights and decrease rapidly as weight increases. The Birmingham stillbirth rates (all weights) are considerably lower than those for New York because the latter include deaths between 20 and 28 weeks, and are based on statistics of 10 years earlier. Table IV shows a sharp rise in stillbirth rates at the highest weights, but in Table III numbers are too small to permit subdivision of births " 9 lb. and over ". The Birmingham rates are based on only 518 stillbirths, and when male and female stillbirth rates are compared at each weight group the results are somewhat irregular (Table III). Even so, in five of the eight weight groups considered, there are no significant differences between the sex-specific stillbirth rates, and it is only at the very low weights that differences are substantial. The New York data exhibited in Table IV are based on much larger numbers, and the

TABLE III

SEX SPECIFIC STILLBIRTH RATES RELATED TO WEIGHT (BIRMINGHAM, 1947)

Birth Weight (lb.)	Stillbirth Rate		Difference (*a*−*b*)
	(*a*) Male	(*b*) Female	
Under 3	326	500	−174±77
3–	402	232	+170±72
4–	144	147	−3±34
5–	53	40	+13±11
6–	16·2	15·6	+0·6±3·5
7–	15	11	+4±3
8–	12	6	+6±3
9 and over	17	10	+7±6
Total ..	24·4	21·0	+3·4±2·0

TABLE IV

SEX SPECIFIC STILLBIRTH RATES RELATED TO WEIGHT (NEW YORK CITY, 1939–1940)

Birth Weight (g.)	Stillbirth Rate		Difference (*a*−*b*)
	(*a*) Male	(*b*) Female	
Under 1,000	664	620	+44±18
1,000–	356	350	+6±21
1,500–	198	161	+37±13
2,000	70	50	+20±5
2,500–	20	14	+6±1
3,000–	12	11·8	+0·2±0·8
3,500–	13·5	13·6	−0·1±1
4,000–	21	27	−6±2
4,500–	57	68	−11±10
5,000 and over	184	180	+4±40
Total ..	34·4	30·0	+4·4±0·8

TABLE V

SEX SPECIFIC STILLBIRTH RATES: NEW YORK DATA GROUPED

Birth Weight (g.)	Total Births			Stillbirths			Stillbirth Rates		Difference (*a*)−(*b*)
	Males	Females	Sex Ratio	Males	Females	Sex Ratio	(*a*) Males	(*b*) Females	
Under 3,500	68,785	75,029	47·8	3,055	2,597	54·1	44·4	34·6	+9·8±1·0
3,500 and over	42,733	29,486	59·2	783	543	59·0	18·3	18·4	−0·1±1·0
Total	111,518	104,515	51·6	3,838	3,140	55·0	34·4	30·0	+4·4±0·8

association between sex-specific stillbirth rates and weight is more consistent. In each weight group below 3,500 g., male rates are higher than female rates; at each weight group above 3,500 g., female rates are higher than male rates (the highest group, which is based on small numbers, excepted).

It is evident that the difference between the sex-specific stillbirth rates (all weights) reflects:

(*a*) differences in rates at each weight group (as given in Tables III and IV)
(*b*) the different weight distribution of male and female total births at risk (as given in Tables I and II).

We can readily assess the effect of (*b*) by standardizing for weight; this we have done for the New York data by applying the observed female sex-specific stillbirth rates in each weight group to the number of male total births in the same weight group. By this procedure, the female stillbirth rate (all weights) is increased from 30·0 to 31·8; that is to say the difference between the sex-specific stillbirth rates is reduced by about two-fifths.

The remainder of the difference in the sex-specific stillbirth rates is accounted for by the higher male rates at the lower weights. The New York data are grouped in Table V, which shows that for births " under 3,500 g." the male stillbirth rate is much higher than the female rate; at "3,500 g. and over" there is no appreciable difference between the sex-specific stillbirth rates.

Table V also gives sex ratios for total births and stillbirths, and enables us to understand the high sex ratio of stillbirths in both weight groups. Although there are more females than males at risk under 3,500 g. (sex ratio 47·8), the sex ratio of stillbirths is raised (54·1) because the male stillbirth rate is much higher than the female rate. At " 3,500 g. and over " there is no difference in the rates, but the considerable excess of males at risk (sex ratio 59·2) again results in a high stillbirth sex ratio (59·0).

It is well known that the sex ratio of stillbirths attributed to difficult labour is high. The New York data do not give cause of stillbirth, but in Birmingham (1945-49) there were 744 stillbirths due to difficult labour, for which the sex ratio was 60·6. It has been suggested that this high ratio may be explained by the fact that male births are on the average heavier than female births (Prinzing, 1907; Pinard and Magnan, 1913; Greulich, 1931). This view is consistent with the evidence here examined, since there are no significant differences between the sex-specific stillbirth rates at the higher weight groups in which difficult labour is the most common cause of death. (Of Birmingham stillbirths at 7 lb. and over, 55 per cent. were attributed to difficult labour.)

Summary

(1) The relation between the sex ratio of stillbirths and birth weight is explored using the following data:

(*i*) for Birmingham, stillbirths notified in the years 1945-49
(*ii*) for New York City, foetal deaths during the two years 1939-40.

(2) The sex ratio of stillbirths is high because:

(*a*) there are more male foetuses at risk (the sex ratio of total births is about 51·5)
(*b*) stillbirth rates are higher for male than for female births.

(3) The sex-specific stillbirth rates are examined in relation to weight at birth. It is shown that male rates are higher than female rates at lower weights (under 3,500 g.), but that for births at higher weights there is no significant difference.

(4) The difference in sex-specific stillbirth rates (all weights) is due:

(*a*) partly to the higher male stillbirth rates at the lower weights

(*b*) partly to sex differences in weight distributions of related total births.

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POLIOMYELITIS IN ENGLAND AND WALES, 1947-1950

BY

W. J. MARTIN

Medical Research Council's Statistical Research Unit, London School of Hygiene and Tropical Medicine

Poliomyelitis has been endemic in England and Wales since its notification was made compulsory in 1912, but prior to 1947 its incidence was, with the exception of three years, of the nature of a sporadic disease with a seasonal rise rather than that of an epidemic disease. This was in contrast to the experience of the United States and some continental countries, where large epidemics had occurred periodically. On the other hand, the vagaries of the disease in England and Wales between the two wars presented some interesting and puzzling features. The fatality rate, i.e. the proportion of deaths to notified cases, varied greatly from year to year; for example, the notifications in 1926 were three times the 1925 level but the fatality rate only half. On the whole, the yearly deaths from poliomyelitis varied only slightly in number, and the concomitant large variations in the notifications would (if they can be accepted as accurate) suggest that the severity of the disease was changing from year to year. Similarly, the incidence of notifications increased more than the mortality during the seasonal period of greater prevalence. This was possibly due to an increased recognition of milder cases at the times when the disease was known to be most likely to occur. The Registrar-General, discussing this possibility, wrote:

> There is evidence that milder cases of the disease are often overlooked, its characteristic after-effects being met with in persons in whom the early stages of the disease have never been recognized.

The only large outbreaks of the disease in England and Wales before the epidemics of 1947-50 occurred in 1926, 1938, and 1940, when 1,159, 1,489, and 1,079 cases were notified. From these occurrences the outbreak of 1947 differed not only in magnitude—708 cases were notified in the week of maximum prevalence—but also in the geographical and age distribution of the patients. This outbreak, in fact, appears to mark a distinct change in the incidence of poliomyelitis in England and Wales, for though a steep decline was recorded in 1948, the incidence remained considerably above the previous endemic level and the year's notifications were larger than those of any year prior to 1947. Any hopes that poliomyelitis would follow a similar trend to that exhibited by cerebrospinal fever after the epidemic of 1941 (when the succeeding outbreaks gradually diminished in size until an endemic level slightly above the original endemic level was reached) were disappointed when large outbreaks of poliomyelitis appeared in 1949 and 1950.

Characteristics of the recent outbreaks have been an apparent change in infectivity and concentration of the disease. It has always been believed that the occurrence of multiple cases of poliomyelitis in a family or even in a household was a very rare event. During the last epidemics two cases in a household were not infrequently reported, and in 1949 three cases of the disease occurred in each of three households in the Isle of Wight (Bradley, 1950). In earlier outbreaks, centres of high incidence generally occurred and there was not much evidence of a spread of infection. Thus in 1926 the neighbouring counties of Leicester, Nottingham, and Warwick contributed 21 per cent. of the total notifications of the country, the population of this area being only 6·7 per cent. of that of the whole country. In 1938, the notifications in London and Essex were 21 per cent. of the total, the population being 14·5 per cent. of the total; in 1940, 34 per cent. of the total cases were notified in Lancashire and Yorkshire West Riding where the population was 13·2 per cent. of that of the whole country. On the other hand, the concentration of the bulk of the cases in a limited area practically disappeared in the recent epidemics, although in 1950 the counties of Warwick, Gloucester, and Stafford had 19 per cent. of the total notifications of the country. The regional distribution for the last 4 years is shown in Table I. The chief features of Table I are the light incidence in 1949 in Wales (particularly North Wales) and in the northern counties, and the high attack rate in 1950 in the west midland counties.

TABLE I
REGIONAL DISTRIBUTIONS

Region	Notifications per Million				Rate as Percentage of Total			
	1947	1948	1949	1950	1947	1948	1949	1950
London	326	61	264	172	148	120	168	82
South Eastern ..	249	51	173	223	113	100	110	106
South Midlands ..	218	73	172	152	99	143	110	72
Eastern	163	49	191	165	74	96	122	79
South Western ..	162	73	211	295	73	143	134	140
West Midlands ..	208	52	111	386	94	102	71	184
North Midlands ..	205	49	163	206	93	96	104	98
North Western ..	209	38	110	129	95	75	70	61
Yorkshire	230	40	188	168	104	78	120	80
Northern	272	29	85	196	123	57	54	93
Wales 1	153	57	96	141	69	112	61	67
Wales 2	173	35	35	238	78	69	22	113
Total England and Wales	221	51	157	210	100	100	100	100

MORTALITY

The age distribution of the deaths from poliomyelitis in 1947 showed a distinct shift compared with earlier outbreaks. The youngest ages formerly supplied the bulk of the deaths, but in 1947 the disease took a much increased toll of the early middle ages. The percentage of deaths under the age of 20 and between the ages of 20 and 45 were 80 and 17 in 1926, 74 and 22 in 1938, and 55 and 41 in 1947.

This change in the age distribution of the deaths was accompanied by an increase in the ratio of male to female deaths. Male deaths expressed as a percentage of female deaths were 118 in 1926, 112 in 1938, and 145 in 1947. The fatality rate fell in the 1947 epidemic, for the notifications in this year were 6·6 and 5·2 times the totals of 1926 and 1938, but the deaths were only 2·9 and 2·7 times as many. The fatality rate rose with age during the more recent outbreaks, the increase being larger for males than for females. The rates are shown in Table II.

TABLE II

DEATHS AS PERCENTAGE OF CORRECTED NOTIFICATIONS, ENGLAND AND WALES

Ages	1947		1948		1949	
	Males	Females	Males	Females	Males	Females
0–	5·8	5·0	2·7	5·5	5·8	6·2
5–	5·2	5·2	10·4	6·2	7·9	6·2
10–	8·0	6·5	8·6	6·7	7·7	7·9
15–	13·7	10·1	25·9	20·1	14·1	18·2
25+	21·8	16·1	32·3	23·6	30·4	23·7

NOTIFICATIONS

The epidemic curves for 1947, 1949, and 1950 varied in shape. In 1947 the notifications rose rapidly from the 23rd week to a maximum of 691 in the 33rd week, declined for two weeks and then, after a further increase to 708, steadily declined to the end of the year. In 1949 the rise to a peak was much slower and more irregular and the maximum incidence of 563 was not reached until the 41st week, from which time there was a continuous decline. In 1950 the outbreak began earlier, but the peak incidence occurred in much the same period as in 1947, the maximum being attained in the 34th week with 589 cases; the subsequent decline was interrupted by occasional small rises. The three epidemics show asymmetrical distributions. Their course is shown in the Figure.

REGIONAL DISTRIBUTION

The notification rates per million for 1947-50 are shown for each county, and for county boroughs, urban districts, and rural districts of each county, in Table III. For England and Wales as a whole there is, it will be seen, very little difference in incidence between the three density areas. Within the counties there are occasional large differences between the types of area, due to intensive outbreaks in particular areas. Thus, in 1947, the rural districts of Surrey had an incidence rate slightly over twice that of the county as a whole, a feature due mainly to an outbreak in Hambledon R.D. with a rate of 1,148 per million. Similarly, the high rate in the urban districts of Bedfordshire in 1947 was mainly due to the unfavourable experience of the town of Luton. Other small areas with a high attack rate in that year included the rural districts of Aysgarth and Bedale in the North Riding of Yorkshire, and Shildon U.D. in the county of Durham. The chief feature of the distribution of the notifications in 1949 was the low incidence in the northern

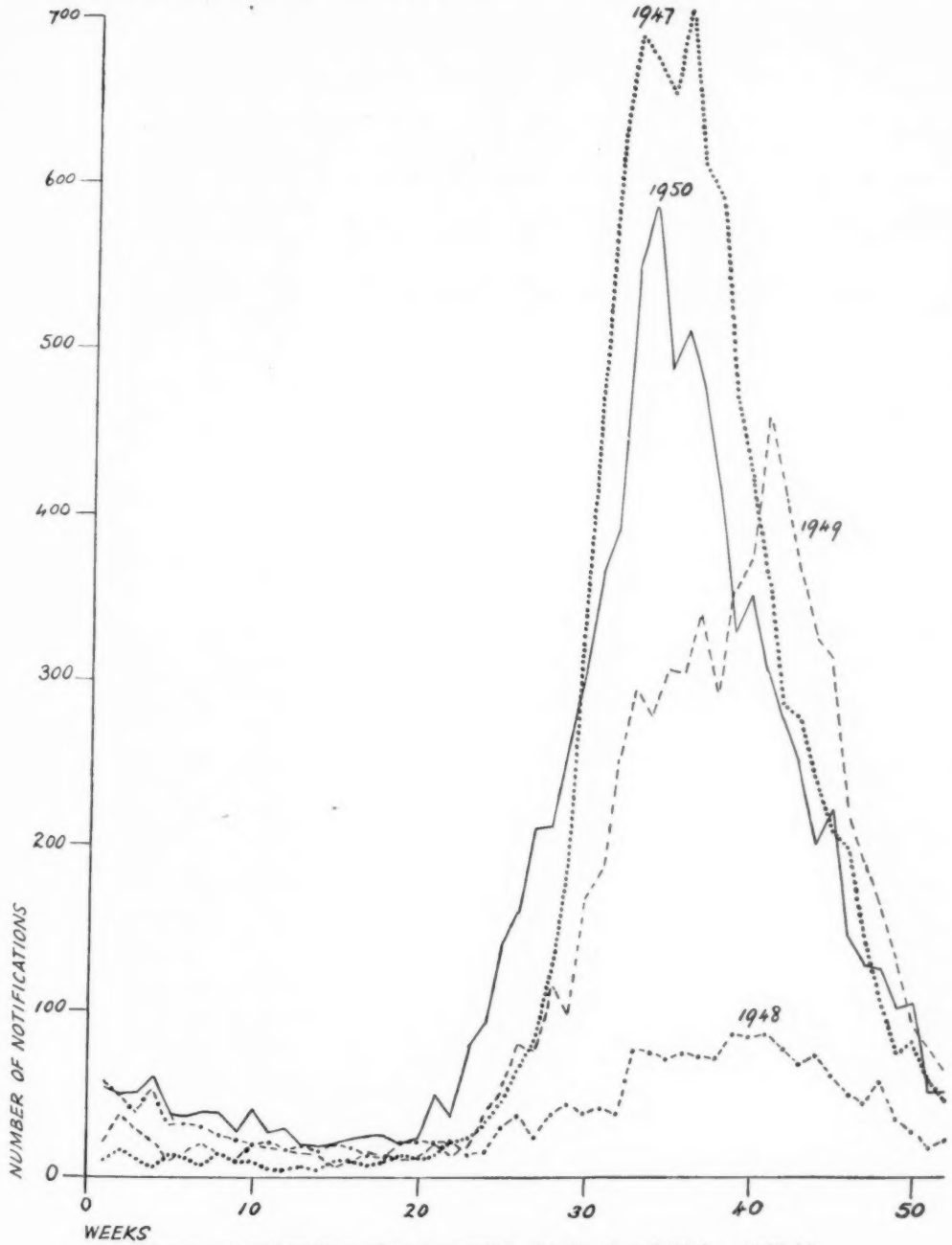

FIGURE.—Notifications of Poliomyelitis, England and Wales, 1947-50.

counties and in Wales, particularly North Wales. The large attack rate in Cornwall was due to an outbreak extending over the districts of Camborne-Redruth U.D., Truro M.B., and Truro R.D., with a combined rate of 780 per million. Bristol had the largest epidemic among the county boroughs, and the experience

TABLE
ACUTE POLIOMYELITIS IN
Notifications

COUNTIES	1947			
	County	C.B.s	U.D.s	R.D.s
LONDON	326			
Surrey	292	318	248	617
Kent	241	123	243	242
Sussex	248	252	214	285
Hants	226	184	238	293
Berks.	174	88	208	212
SOUTH EASTERN	249	217	240	304
Middlesex	237		237	
Hertford	236		233	245
Bucks.	130		136	125
Oxford	212	212	242	200
Northants	133	224	56	171
Hunts.	149		214	92
Bedford	386		458	217
Cambridge	93		103	81
SOUTH MIDLANDS	218	218	230	168
Essex	156	102	166	207
Suffolk	170	60	218	195
Norfolk	182	263	203	128
EASTERN	163	134	172	171
Wilts.	198		129	267
Dorset	289		256	347
Devon	112	86	124	128
Cornwall	138		168	102
Somerset	161	235	110	186
SOUTH WESTERN	162	120	152	190
Gloucester	216	221	164	235
Hereford	398		302	465
Salop	140		142	139
Staffs.	151	156	136	177
Worcester	160	90	140	284
Warwick	265	257	311	244
WEST MIDLANDS	208	215	180	234
Leicester	232	386	103	103
Rutland	158		279	130
Lincs.	336	348	247	402
Notts.	120	65	140	182
Derby	162	158	177	146
NORTH MIDLANDS	205	234	169	219

of this city adversely affected the rate for Gloucestershire. In 1950 there was a definite peak of cases in the west midlands. Within this area the highest rates

III
ENGLAND AND WALES, 1947-1950
per 1,000,000

1948				1949				1950			
County	C.B.s	U.D.s	R.D.s	County	C.B.s	U.D.s	R.D.s	County	C.B.s	U.D.s	R.D.s
61				264				172			
55	53	54	70	129	68	138	163	152	156	137	272
58	79	58	55	208	113	198	254	204	302	213	159
36	11	32	64	136	155	107	151	134	83	170	141
47	49	35	61	184	170	189	206	425	289	638	362
56	44	75	54	259	329	251	221	136	69	115	185
51	41	51	60	173	159	167	202	223	195	235	218
69		69		179		179		140		140	
63		54	87	215		189	282	151		147	162
97		121	72	136		129	142	172		161	184
60	86	21	53	153	186	125	135	124	93	209	119
94	67	126	71	153	105	188	155	284	153	350	294
0		0	0	209		34	346	90		68	106
97		124	33	189		123	332	146		161	114
63		94	26	91		138	34	68		62	76
73	76	76	58	172	146	171	185	152	123	151	161
43	50	41	41	207	121	218	310	153	123	160	166
28	0	44	32	142	58	153	179	71	58	66	82
87	137	71	61	171	159	154	183	286	201	308	331
49	63	43	47	191	121	209	222	165	132	159	211
121		138	103	205		201	207	205		196	212
77		82	69	255		254	256	430		370	530
47	49	38	54	149	106	149	200	304	201	359	363
57		49	67	340		454	202	287		315	254
85	90	90	79	201	272	165	212	281	155	298	304
73	59	76	74	211	141	235	211	295	191	312	317
70	94	42	40	255	380	168	77	468	605	343	286
40		39	41	181		235	145	315		215	382
55		29	82	41		50	33	220		236	206
39	42	38	30	51	53	54	38	291	213	377	311
29	24	33	27	44	56	47	25	502	769	449	346
61	68	39	43	115	104	153	134	425	472	322	234
52	64	37	43	111	139	88	71	386	438	358	285
57	71	13	73	226	233	292	158	255	367	118	201
156		277	128	231		538	167	369		269	390
50	31	34	74	187	187	234	150	352	293	225	482
44	27	55	53	186	196	182	176	149	176	112	183
46	70	34	48	71	35	45	116	100	139	81	103
49	49	38	63	163	180	166	145	206	252	127	256

were found in the county boroughs, and the towns most affected were Dudley, Bristol, and Birmingham, where the rates were 1,050, 691, and 507 per million.

TABLE

Counties	1947			
	County	C.B.s	U.D.s	R.D.s
Cheshire	190	146	178	342
Lancashire	214	213	215	221
North Western	209	205	205	266
West Riding	224	187	268	284
East Riding	290	267	325	321
North Riding	206	136	92	421
Yorkshire	230	194	248	322
Durham	285	220	333	310
Northumberland	231	202	239	305
Cumberland	301	108	242	443
Westmorland	367		258	466
Northern	272	206	294	350
Brecknock	282		254	294
Carmarthen	291		366	236
Glamorgan	147	129	134	227
Monmouth	97	109	67	260
Wales 1	153	125	134	240
Caernarvon	89		126	39
Cardigan	228		338	172
Denbigh	145		171	123
Flint	274		376	167
Merioneth	130		58	188
Montgomery	335		116	473
Pembroke	118		123	114
Radnor	356		320	372
Anglesey	42		0	68
Wales 2	173		192	156
Total	221	199	211	239

The distribution of poliomyelitis in some of these localized outbreaks has been quite different from that exhibited by other infectious diseases. Gilloran (1949), investigating the outbreak in Dorset in 1947, found that the better social classes had the highest attack rates, and that in the borough of Poole where the incidence was high the majority of the cases occurred in the better-class residential districts. A similar phenomenon was reported for Luton (Grundy, 1948), where poliomyelitis occurred "in circumstances of good health and low infection—a characteristic of poliomyelitis which seems to favour populations in favourable conditions where the public health services are above the average". Outbreaks in other areas did not show this social distinction. Daley and Benjamin (1948) found an insignificant correlation between the proportion of males aged 14+ who were in social classes IV and V and the attack rates of poliomyelitis in the 28 London boroughs. Hill and

III—*continued*

1948				1949				1950			
County	C.B.s	U.D.s	R.D.s	County	C.B.s	U.D.s	R.D.s	County	C.B.s	U.D.s	R.D.s
43	47	34	70	114	56	128	204	120	132	123	83
37	40	31	35	109	97	131	103	131	155	96	87
38	41	32	48	110	92	131	141	129	152	103	85
40	35	42	54	220	237	209	171	171	190	123	211
54	65	39	39	91	88	77	113	107	105	126	94
28	63	15	13	63	34	39	115	207	172	187	258
40	41	38	42	188	206	176	148	168	179	132	205
32	23	44	22	45	52	37	49	179	171	194	162
21	28	15	20	124	177	92	40	262	293	194	378
40	46	36	40	135	89	59	209	135	149	118	140
15		32	0	286		189	375	45		31	58
29	26	34	24	85	100	61	106	196	214	183	190
56		63	53	89		234	26	107		176	77
94		153	51	76		85	70	216		310	150
63	85	47	57	103	114	110	60	154	131	131	272
26	29	22	46	85	9	105	138	78	85	76	69
57	75	48	54	96	95	109	68	141	123	129	197
24		42	0	24		28	19	210		183	247
19		0	28	37		108	0	56		0	85
6		0	11	12		26	0	385		348	416
51		43	59	83		99	68	194		141	245
128		0	229	0		0	0	122		57	171
66		56	36	66		0	108	502		669	395
35		48	22	23		24	22	124		118	130
49		0	72	99		154	72	444		463	435
20		0	33	0		0	0	100		104	98
35		27	42	35		44	28	238		214	258
51	48	50	53	157	138	153	151	210	228	186	226

Martin (1949) found no relation between the attack rate of poliomyelitis and the crude death rate from all causes, nor between the attack rate and the infant mortality rate in the 126 large towns and 148 smaller towns, nor between the attack rate of poliomyelitis and the overcrowding index in the London boroughs. Table III shows that, in general, the attack rate in a county is very similar in each density area. In 1950 the correlation coefficient between the attack rates in the urban and rural districts of the counties of England and Wales was 0·75.

Some Epidemiological Factors

The spread of infection presents some interesting problems, since poliomyelitis, except in the more advanced stage of the disease, cannot be diagnosed with certainty and many minor attacks probably escape notice or do not require medical attention.

Although in England and Wales the incidence of poliomyelitis has always begun to rise with the advent of warm weather and has reached epidemic proportions in the summer, its relationship to weather conditions is not very definite. The hot dry summer of 1949 might be held to favour the theory of a dust-borne infection, but the larger outbreak in the following year occurred in a wet cold summer.

An important contribution to knowledge of the incidence of the disease in the child population has been made by Casey and others (1950). In a field investigation carried out in Chicago, they showed that subclinical attacks in children were common. Poliomyelitis virus was recovered from the stools of 53 per cent. of contacts (i.e. children living in the immediate neighbourhood of a paralytic case), from 11 per cent. of non-contacts (i.e. children living in the surrounding neighbourhood of the first group), and from 8 per cent. of controls (i.e. children living ten blocks away from the patient with a clinical attack). The authors estimate that probably most children in Chicago had had one subclinical attack of poliomyelitis by their sixth birthday—either different strains or reinfections—and that among Chicago children paralysis was a condition that occurred less than once in 200 cases. We know that trauma can, at least sometimes, affect the course of the disease, for McCloskey (1950) found in Victoria, Australia, that a relation existed in a number of cases between an injection of an immunizing agent and the subsequent development of paralytic poliomyelitis. Hill and Knowelden (1950) found that the distribution of the bodily sites of paralysis was quite abnormal in children who had been inoculated within the month preceding the onset of their illness. In children recently inoculated, the injected limb (arm or leg) was much more frequently a site of paralysis than in children not recently inoculated. Hill and Knowelden concluded " that there may be present in the poliomyelitis group cases which would not have been clinically diagnosed as poliomyelitis at all if their inoculation had not brought them into the paralytic group ". The finding of Casey and his co-workers that poliomyelitis is generally a non-clinical condition from which clinical cases only rarely develop, is in agreement with many of the observed features of the trend of the disease. This would explain the rarity of multiple cases in a family, which has been generally noted in former epidemics, and the larger incidence in the better residential districts, which has been occasionally observed in some local outbreaks. It is well known that with infectious diseases of childhood the age of attack is correlated with density, i.e. children living in overcrowded conditions contract infectious diseases, on the average, at younger ages than their more fortunate brethren living in a better environment. This may well be the case with poliomyelitis; children living in the less congested district of an area may have been subjected to a much smaller risk of a subclinical attack than the remainder, and are consequently much more likely to develop a clinical attack during an outbreak.

The factors which determine whether paralysis develops or not have not been found. The evidence that inoculation can have this effect is fairly conclusive, but this factor can account for only an exceedingly small proportion of the total cases. Exercise in the pre-paralytic stage also appears to be of importance. It has been

shown by several workers that the usual vital statistical indices of general living conditions are unrelated to the level of the incidence rate. Table III does not suggest that any particular area has a peculiar liability to a high incidence rate, nor that a high incidence in one outbreak is necessarily followed by a low incidence in a succeeding outbreak. To examine the latter point in more detail the attack rates in the 28 London boroughs in successive outbreaks were correlated, and the correlations were $0{\cdot}273 \pm 0{\cdot}175$ for 1947 and 1949, and $0{\cdot}077 \pm 0{\cdot}188$ for 1949 and 1950.

Both correlation coefficients are insignificant, and it may therefore be stated that in these areas there was no relation between successive outbreaks.

It also seemed of interest to examine whether the variations in the incidence of acute poliomyelitis between areas were related to the prevalence of other infectious diseases. Accordingly, the notification rates of scarlet fever, whooping cough, diphtheria, and measles in the 28 London boroughs were correlated separately and in total with the notification rates of acute poliomyelitis. (See Table IV.)

TABLE IV

CORRELATIONS BETWEEN ATTACK RATE FROM POLIOMYELITIS AND OTHER INFECTIOUS DISEASES

Year		1947	1948	1949	1950
		r	*r*	*r*	*r*
Poliomyelitis and	scarlet fever	$0{\cdot}406 \pm 0{\cdot}16$	$0{\cdot}051 \pm 0{\cdot}18$	$-0{\cdot}155 \pm 0{\cdot}18$	$0{\cdot}522 \pm 0{\cdot}14$
	whooping cough	$0{\cdot}175 \pm 0{\cdot}18$	$0{\cdot}259 \pm 0{\cdot}18$	$0{\cdot}093 \pm 0{\cdot}18$	$0{\cdot}027 \pm 0{\cdot}19$
	diphtheria	$0{\cdot}735 \pm 0{\cdot}09$	$0{\cdot}428 \pm 0{\cdot}15$	$0{\cdot}092 \pm 0{\cdot}19$	$0{\cdot}040 \pm 0{\cdot}19$
	measles	$-0{\cdot}087 \pm 0{\cdot}19$	$0{\cdot}053 \pm 0{\cdot}19$	$-0{\cdot}006 \pm 0{\cdot}19$	$0{\cdot}319 \pm 0{\cdot}17$
	combined rate of these four diseases	$0{\cdot}163 \pm 0{\cdot}18$	$0{\cdot}134 \pm 0{\cdot}19$	$-0{\cdot}002 \pm 0{\cdot}19$	$0{\cdot}324 \pm 0{\cdot}17$

The incidence of whooping cough and measles was unrelated to the incidence of poliomyelitis in each year, and the distribution of poliomyelitis followed the distribution of diphtheria in 1947 and 1948 and that of scarlet fever in 1947 and 1950. The combined incidence of the four infectious diseases showed no relation to poliomyelitis in any of the four years. Thus it appears that the incidence of poliomyelitis was unrelated to that of the common infectious diseases of childhood.

SUMMARY

The epidemic curve of poliomyelitis in England and Wales during 1947-50 showed considerable annual fluctuations.

In 1947 and 1950 the incidence rose steadily to a peak, but in 1949 the rise was not continuous and the maximum occurred several weeks later. In 1950 the rise in incidence began about a month earlier.

For England and Wales as a whole there is no evidence that the towns have a greater liability than the rural districts. The disease was more widely scattered in 1950 than in former years of high incidence, although in 1949 the northern counties and Wales had a relatively light experience.

The area incidence of successive outbreaks did not appear to be related.

In the London boroughs the incidence was not related to that of infectious diseases of childhood.

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BIRTH WEIGHTS OF SOUTH AFRICAN BABIES
(II.) EFFECT OF BIRTH RANK ON BIRTH WEIGHT

BY

EVA J. SALBER* and EVELYN S. BRADSHAW

Institute of Family and Community Health, Union Health Department, Durban, Union of South Africa

This paper is a report on the birth weights of groups of South African babies with special reference to the effect of birth rank on these weights. This is the second paper of a series, the first of which (Salber and Bradshaw, 1951) dealt with the effect of race and sex on birth weight.

Material and Method

The data were collected from various hospitals and nursing homes in Durban, Pietermaritzburg, and Capetown, as shown below:

3,165 European babies from two private nursing homes in Durban.

1,057 coloured (i.e. half-caste)† babies from a mission hospital in Durban, and two provincial hospitals, one in Capetown and one in Pietermaritzburg.

2,188 Bantu babies from a mission hospital in Durban.

1,391 Indian babies from a provincial hospital in Pietermaritzburg, a mission hospital in Durban, and a government-aided hospital in Durban.

The data extracted in all cases consisted of birth weight, race of mother, sex of child, and birth rank of child. Abortions and multiple births were excluded. The numbers of cases used here are not identical with those used in the previous paper, since those in which birth rank was not recorded had to be omitted. Additional data which included birth rank was collected for this paper. In considering the birth rank of the child, previous miscarriages were included.

Frequency distributions of the birth ranks for the different racial groups were drawn up (see Appendix, pp. 252-53). Means and standard deviations of the birth weights for the different birth ranks were calculated for the races and sexes separately. Where differences occurred the significance of these differences was also calculated.

Results

Table I (overleaf) shows that over 80 per cent. of the European babies fall into the first two birth ranks, whereas for the Indian babies 80 per cent. covers the first five birth ranks, and over 5 per cent. of the Indian babies are of the tenth or higher birth ranks. The Coloured and Bantu babies fall between these two

* Senior Bursar, Council for Scientific and Industrial Research.

† Coloured people are persons of mixed race, at least one of whose progenitors is a European.

TABLE I

PERCENTAGE DISTRIBUTION BY BIRTH RANK (BOTH SEXES)

Birth Rank	European	Coloured	Bantu	Indian
1	49·0	37·3	32·0	29·0
2	32·0	24·1	25·7	18·8
3	13·6	12·1	16·5	12·6
4	3·2	7·5	9·4	9·3
5	1·3	4·7	6·4	8·0
6 and 7	0·6	7·4	6·4	10·6
8 and 9	0·2	3·8	2·2	6·6
10+	0·1	3·1	1·4	5·1
Total	100·0	100·0	100·0	100·0

extremes, with the Coloured babies showing slight predominance over the Bantu in the higher birth ranks.

Table II and the Figure show that the mean birth weight increases with increasing birth rank up to Rank 4 in all the racial groups, although in no group is there a steady rise from Rank 1 to the highest rank. The European and Indian groups rise to a peak (which lasts longer in the Indian group) and the mean birth weights fall for the highest birth rank group, although remaining higher than Rank 1 babies. In the Coloured group, with the exception of the slight drop in mean birth weight for Ranks 6 and 7, the curve is very similar to that found in the European and Indian groups. The Bantu curve, however, seems to differ from the other three, in that there is a drop after Rank 4, carried on to Ranks 6 and 7, after which the mean birth weights rise again to a peak at Rank 10+. It may be that this is a question of numbers, as none of the differences between mean birth weights for the higher birth ranks are significant.

Although the mean birth weights follow the patterns previously described, the only significant differences found were between Rank 1 and the other ranks (Table II), with two exceptions in which Rank 2 babies were also significantly lower than two of the higher ranks.

It has been shown (Salber and Bradshaw, 1951) that significant differences existed between the mean birth weights of the four racial groups, with the exception of that between Coloured and Bantu babies. In the light of the very different distribution of ranks found in the four racial groups (see Table I), this was considered to be a possible factor in accounting for the difference found between mean birth weights. Accordingly, like ranks were compared with each other in the four racial groups, and as the sex ratio appeared to vary in the different ranks, boys and girls were considered separately (see Table III, overleaf).

Table III shows that for both sexes and for all ranks, the same differences exist as those found previously. That is to say that, in all ranks, European babies are significantly higher, Indians are significantly lower, and there is no significant difference between the Coloured and Bantu groups.

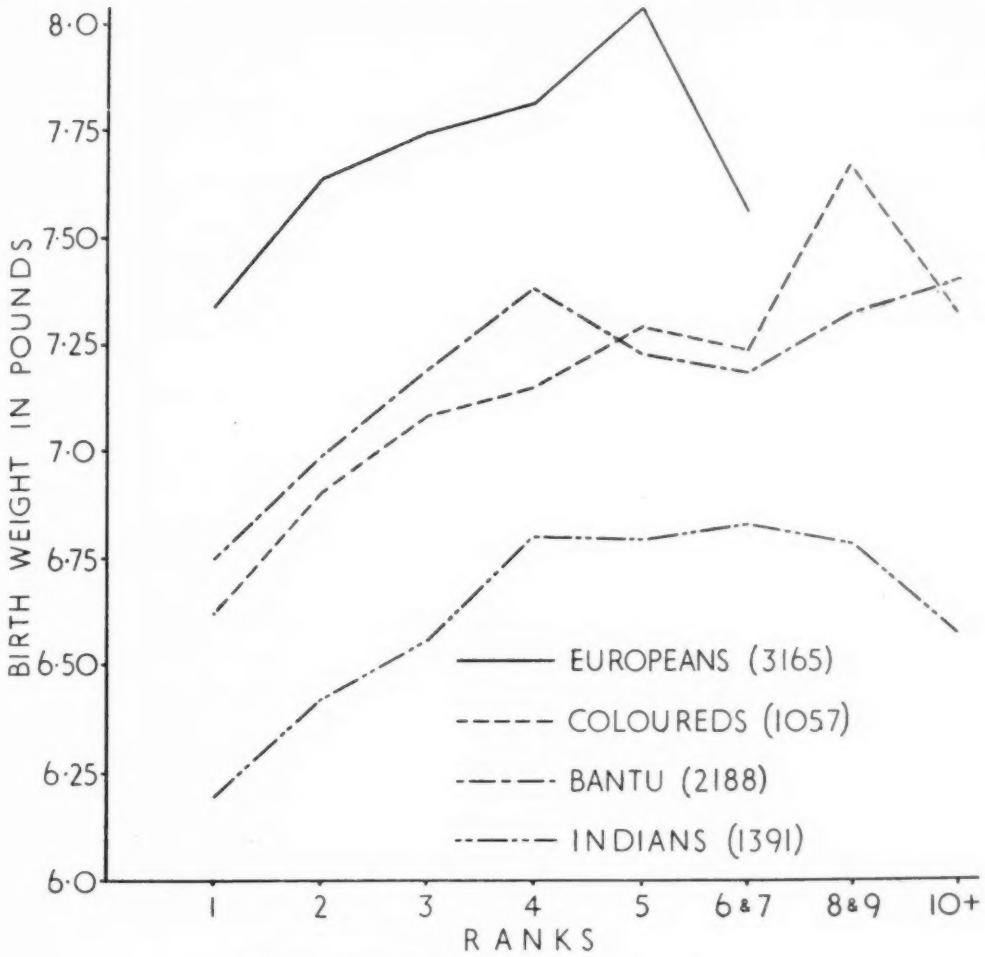

FIGURE.—Birth weight according to birth rank.

TABLE II

MEAN BIRTH WEIGHT RELATED TO BIRTH RANK

Birth Rank	European			Coloured			Bantu			Indian		
	No. of Births	Mean (lb.)	S.D.	No. of Births	Mean (lb.)	S.D.	No. of Births	Mean (lb.)	S.D.	No. of Births	Mean (lb.)	S.D.
1	1,551	7·34	1·13	394	6·63	1·09	701	6·75	0·98	404	6·20	0·99
2	1,013	7·64	1·14	255	6·90	1·12	562	6·99	1·13	261	6·43	1·11
3	430	7·74	1·15	128	7·08	1·08	360	7·19	1·07	175	6·55	0·96
4	101	7·81	1·31	80	7·15	1·48	206	7·38	1·29	129	6·79	1·16
5	40	8·03	1·26	50	7·29	1·03	141	7·23	1·15	112	6·79	1·32
6 and 7	21	7·56	1·26	78	7·24	1·70	140	7·18	1·38	147	6·82	1·49
8 and 9	7	—	—	40	7·68	1·20	48	7·33	1·09	92	6·76	1·32
10 and over	2	—	—	32	7·33	1·63	30	7·40	1·01	71	6·58	1·54
Total	3,165	7·52	1·16	1,057	6·93	1·23	2,188	7·03	1·11	1,391	6·51	1·19

TABLE III

MEAN BIRTH WEIGHT FOR EACH SEX RELATED TO BIRTH RANK

Birth Rank	Mean (lb.) Male				Mean (lb.) Female			
	European	Coloured	Bantu	Indian	European	Coloured	Bantu	Indian
1	7·43	6·67	6·85	6·22	7·21	6·55	6·63	6·16
2	7·73	6·86	7·13	6·37	7·52	6·90	6·81	6·47
3	7·87	7·27	7·28	6·78	7·63	6·92	7·09	6·24
4 and over	7·92	7·48	7·44	6·88	7·79	7·10	7·12	6·65

$P<0·01$ for differences between means of all racial groups except:
MALE: Coloured and Bantu (all birth ranks).
FEMALE: Coloured and Bantu (all birth ranks).
European and Coloured (4 and over. $P<0·05$).

DISCUSSION

Meredith and Brown (1939), in a review of the literature on the relation between birth order and mean weight at birth, found that all investigators agreed that later-born babies exceeded firstborn babies in mean birth weight. There does not seem to be unanimity, however, as to whether the birth weight becomes progressively greater with each increasing birth rank. One of the difficulties encountered by most investigators, including ourselves, is the paucity of numbers in the higher birth ranks. For this reason many investigators group their birth ranks.

Meredith and Brown divided their cases into firstborn, second- to fourth-born, and fifth and higher ranks. Of these three groups, they found that the second- to fourth-born had a higher mean birth weight than the firstborn; and that the fifth- and later-born had a higher mean birth weight than the second- to fourth-born. They also quote Perlstein and Levinson (1937), who found that birth weight increases with the parity of the mother up to the seventh, after which it decreases.

Bakwin and Bakwin (1934) state that birth weight increases up to the sixth pregnancy, after which it declines. Martin (1931) gave results for his mean birth weights for individual ranks up to 16, and found that

> the weight of the later-born exceeds that of the firstborn but it seems doubtful whether weight follows a progression with the rank of birth.

Donald (1939) quotes other workers in this field, and concludes that

> most investigators agree that there is an increase in weight up to the third child. What happens after that has not been satisfactorily determined owing to the inadequacy of the available numbers, but it seems likely that there is but slight change from the fourth onwards.

McKeown and Gibson (1951) found that weight increases with parity to the third birth rank, but above the third there is no consistent change (at least none is evident when Rank 3 is compared with Rank 4 and over).

In this series, the results were different for the different racial groups, except for the fact that all groups showed an increase in mean birth weight up to Rank 4. The Europeans showed their peak birth weight at Rank 5, with a drop thereafter. The Coloureds showed an almost steady rise to a peak at Ranks 8 and 9, followed by a drop. The Indians rose to a peak at Rank 4, remained virtually stationary to Ranks 8 and 9, and then dropped. The Bantu rose to a peak at Rank 4, dropped slightly for Ranks 5, 6, and 7, and rose again to a second peak at Rank 10+.

We have observed previously (see Table I) that the percentage distribution of ranks found was different for the racial groups. It is possible that the age distribution of the mothers in the different ranks is also likely to differ in the racial groups. Whether this may account for some of the differences found between the four groups is not yet known, and is in the process of investigation.

Summary

Birth weights of infants were studied with special reference to birth rank. The material comprised 3,165 European babies, 1,057 Coloured babies, 2,188 Bantu babies, and 1,391 Indian babies. Results were as follows:

(1) Distribution of birth ranks. Far more babies of higher birth rank were found in the Coloured, Bantu, and Indian groups than in the European group, with 5 per cent. of Indian babies of 10th or higher birth rank.

(2) Mean birth weight increases with increasing birth rank up to Rank 4 in all racial groups. In no case is there a steady progression up to the highest rank. After Rank 4 the pattern is not consistent for the various groups. The only significant differences found were between Rank 1 and the higher ranks.

(3) The racial differences in mean birth weights which were noted in our previous paper are not due to differences in birth rank distribution of the babies in each race group. The present report indicates that these differences are to be found when comparing the various groups of babies of the same birth rank.

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APPE
DISTRIBUTION OF BIRTH

Birth Weight (lb.)	Birth Rank										
	1		2		3		4 and Over		All Birth Ranks		
	M	F	M	F	M	F	M	F	M	F	Total
	(a) European										
1 –	—	—	—	—	—	—	—	—	—	—	—
1½–	—	—	—	—	—	—	1	—	1	—	1
2 –	1	—	1	—	—	—	—	—	2	—	2
2½–	—	2	1	—	—	—	—	—	1	2	3
3 –	3	3	2	—	—	1	—	—	5	4	9
3½–	—	1	1	—	2	—	—	—	3	1	4
4 –	7	3	2	2	—	—	—	1	9	6	15
4½–	8	11	3	3	1	1	1	2	13	17	30
5 –	20	16	6	12	4	2	—	—	30	30	60
5½–	34	48	15	15	9	7	2	2	60	72	132
6 –	81	87	36	42	10	21	7	7	134	157	291
6½–	107	128	50	82	19	23	4	10	180	243	423
7 –	159	141	63	99	32	47	14	10	268	297	565
7½–	124	128	113	93	31	42	22	13	290	276	566
8 –	149	90	87	80	42	27	9	14	287	211	498
8½–	62	45	59	42	26	27	13	10	160	124	284
9 –	37	20	36	28	16	14	8	6	97	68	165
9½–	16	6	16	7	11	6	3	4	46	23	69
10 –	9	3	5	4	7	1	3	1	24	9	33
10½–	1	1	1	4	—	—	1	2	3	7	10
11 –	—	—	1	1	1	—	1	—	3	1	4
11½–	—	—	—	—	—	—	—	—	—	—	—
12 –	—	—	—	—	—	—	—	—	—	—	—
12½–	—	—	—	—	—	—	—	—	—	—	—
13 –	—	—	1	—	—	—	—	—	1	—	1
Total	818	733	499	514	211	219	89	82	1,617	1,548	3,165
	(b) Coloured										
2 –	1	1	—	—	—	—	1	—	2	1	3
2½–	—	—	—	—	—	—	1	—	1	—	1
3 –	1	1	—	1	—	1	2	1	3	4	7
3½–	1	1	—	1	—	—	2	3	3	5	8
4 –	5	5	1	2	1	1	3	3	10	11	21
4½–	4	2	4	—	—	—	—	1	8	3	11
5 –	12	14	7	6	2	2	1	—	22	22	44
5½–	27	19	21	11	4	9	6	14	58	53	111
6 –	35	48	21	21	6	10	11	20	72	99	171
6½–	46	40	21	17	9	11	21	26	97	94	191
7 –	25	23	18	30	14	16	15	22	72	91	163
7½–	21	24	20	13	8	8	29	20	79	65	144
8 –	12	10	12	10	10	3	17	15	51	38	89
8½–	11	2	5	5	7	3	10	8	33	18	51
9 –	3	—	1	3	—	3	9	1	13	7	20
9½–	—	—	3	1	—	—	5	2	8	3	11
10 –	—	—	—	—	—	—	4	4	4	4	8
10½–	—	—	—	—	—	—	—	1	—	1	1
11 –	—	—	—	—	—	—	1	—	1	—	1
11½–	—	—	—	—	—	—	—	—	—	—	—
12 –	—	—	—	—	—	—	1	—	1	—	1
Total	204	190	134	121	61	67	139	141	538	519	1,057

NDIX

WEIGHTS AT EACH BIRTH RANK

Birth Weight (lb.)	Birth Rank										
	1		2		3		4 and Over		All Birth Ranks		
	M	F	M	F	M	F	M	F	M	F	Total
	(*c*) *Bantu*										
1½–	—	—	1	1	—	—	1	—	2	1	3
2 –	—	2	1	—	2	—	2	—	5	2	7
2½–	1	—	2	—	—	—	1	—	4	—	4
3 –	—	1	—	2	—	—	1	1	1	4	5
3½–	2	2	1	—	1	—	2	4	6	6	12
4 –	3	1	4	4	1	2	4	2	12	9	21
4½–	8	6	3	4	—	1	—	3	11	14	25
5 –	12	17	12	11	4	5	7	8	35	41	76
5½–	28	42	6	18	11	8	10	16	55	84	139
6 –	48	92	34	50	27	25	23	35	132	202	334
6½–	93	80	49	74	24	33	46	49	212	236	448
7 –	68	55	66	51	49	32	54	48	237	186	423
7½–	44	36	39	47	37	24	55	47	175	154	329
8 –	20	16	28	15	28	12	39	20	115	63	178
8½–	12	5	17	7	12	9	34	16	75	37	112
9 –	1	3	7	1	5	4	15	8	28	16	44
9½–	2	—	4	1	2	—	4	2	12	3	15
10 –	1	—	1	—	1	—	3	3	6	3	9
10½–	—	—	1	—	1	—	1	—	3	—	3
11 –	—	—	—	—	—	—	1	—	1	—	1
Total	343	358	276	286	205	155	303	262	1,127	1,061	2,188

	(*d*) *Indian*										
1 –	—	—	—	—	—	—	1	2	1	2	3
1½–	—	—	1	—	—	—	—	—	1	—	1
2 –	—	—	—	—	—	—	2	—	2	—	2
2½–	2	1	—	—	—	—	1	1	3	2	5
3 –	1	1	4	—	—	—	2	5	7	6	13
3½–	—	1	3	1	—	—	4	3	7	5	12
4 –	3	8	4	—	1	2	1	7	9	17	26
4½–	7	13	1	1	1	6	7	9	16	29	45
5 –	34	28	11	18	7	10	15	16	67	72	139
5½–	27	36	19	19	11	9	25	20	82	84	166
6 –	46	41	30	30	17	20	38	58	131	149	280
6½–	30	38	17	19	22	12	49	38	118	107	225
7 –	31	28	24	24	20	8	48	51	123	111	234
7½–	8	7	11	5	16	3	39	34	74	49	123
8 –	4	6	7	4	6	1	18	12	35	23	58
8½–	2	—	2	4	1	1	16	6	21	11	32
9 –	—	1	1	1	—	1	6	6	7	9	16
9½–	—	—	—	—	—	—	1	2	1	2	3
10 –	—	—	—	—	—	—	2	4	2	4	6
10½–	—	—	—	—	—	—	2	—	2	—	2
Total	195	209	135	126	102	73	277	274	709	682	1,391

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SECULAR CHANGES IN THE INCIDENCE OF MALFORMATIONS OF THE CENTRAL NERVOUS SYSTEM

BY

BRIAN MacMAHON*, R. G. RECORD, and THOMAS McKEOWN

From the Department of Social Medicine, University of Birmingham

In a previous communication (Record and McKeown, 1949) it was noted that in Birmingham the incidence of anencephalus and spina bifida decreased during the years 1940-47, while the incidence of hydrocephalus remained fairly constant. It was tentatively suggested that this decrease might be correlated with changes in the parity distribution of births, since it was shown that the incidence of anencephalus and spina bifida is associated with birth order. We propose here to examine this suggestion more critically, making use of statistics for additional years and of more detailed information than was previously available about the parity distribution of the general population of births.

Table I (see Fig. 1) gives the incidence of malformations of the central nervous system notified as stillbirths or infant deaths in Birmingham for the period 1936-49. Anencephalus associated with spina bifida is classified under anencephalus, and spina bifida with hydrocephalus under spina bifida. During the years 1940-49 the incidence of anencephalus and spina bifida fell sharply; the incidence of hydrocephalus showed no consistent variation. The significance of these changes

* In receipt of a personal grant from the Medical Research Council.

TABLE I

INCIDENCE (PER THOUSAND TOTAL BIRTHS) OF MALFORMATIONS OF THE CENTRAL NERVOUS SYSTEM
Birmingham, 1936-49

Years	Malformations		
	Anencephalus	Spina bifida	Hydrocephalus
1936 and 1937	2·0 (69)	2·6 (91)	0·9 (31)
1938 and 1939	2·5 (90)	2·4 (88)	1·4 (51)
1940 and 1941	2·8 (87)	3·0 (93)	1·1 (33)
1942 and 1943	2·7 (101)	2·9 (112)	0·9 (35)
1944 and 1945	2·2 (93)	2·2 (92)	0·7 (31)
1946 and 1947	1·8 (85)	1·9 (92)	1·1 (51)
1948 and 1949	1·6 (65)	1·7 (71)	0·7 (30)
Total	2·18 (590)	2·37 (639)	0·97 (262)
1940–1944 (*a*)	2·58 (236)	2·84 (259)	0·93 (85)
1945–1949 (*b*)	1·80 (195)	1·86 (201)	0·88 (95)
Difference (*a*)—(*b*)	0·78±0·21	0·98±0·22	0·05±0·14

Numbers in brackets are the numbers of malformations (stillbirths and infant deaths).

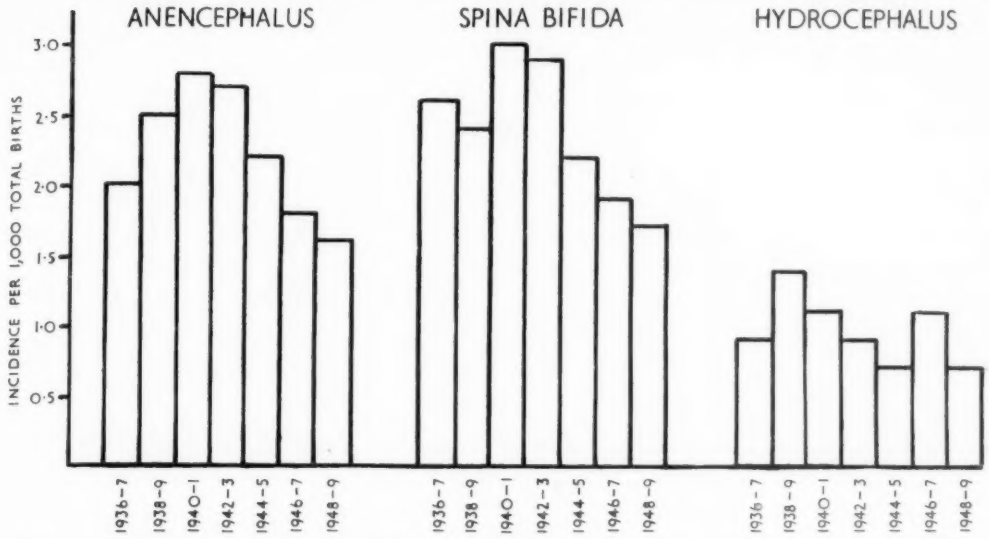

FIG. 1.—Incidence of malformations of the central nervous system. Birmingham, 1936–49.

is indicated by a comparison of the rates attributed to each malformation in the periods 1940-44 and 1945-49.

Before enquiring whether this variation is correlated with changes in the parity structure of the related population of births, we may reconsider and extend slightly the evidence of association of incidence with birth order. This association, previously demonstrated for anencephalus and spina bifida (but not for hydrocephalus) on Birmingham data, is also exhibited for anencephalus* on Scottish data in Table II (see Fig. 2). There is good

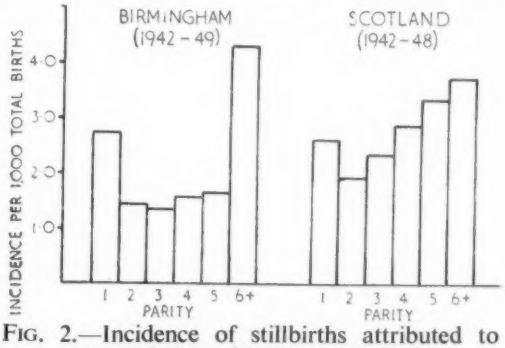

FIG. 2.—Incidence of stillbirths attributed to anencephalus related to birth rank.

* Since nearly all anencephalics are stillborn, the relationship between incidence and birth rank can be examined on Scottish statistics for stillbirths.

TABLE II

INCIDENCE OF ANENCEPHALUS RELATED TO BIRTH RANK

Birth Rank	Incidence per 1,000 Total Births	
	Birmingham 1942–1949 (stillbirths and infant deaths)*	Scotland 1942–1948 (stillbirths only)
1	2·75	2·64
2	1·42	1·95
3	1·31	2·36
4	1·53	2·88
5	1·64	3·37
6 and over	4·32	3·73

* Of the total, 95 per cent. are stillbirths.

agreement in the figures for Birmingham and Scotland in birth rank 1 and in birth rank 6 *and over*; but in Scotland incidence rises more sharply after birth rank 2, and is actually higher in birth ranks 4 and 5 than in 1. These observations confirm the association of incidence with parity, but suggest that the risk of anencephalus is not invariably greater in the first than in the later birth ranks. This modifies somewhat the generality of the conclusion (suggested by Birmingham data) as to the importance of the proportion of first born in determining the incidence of anencephalus in a population of births.

To decide whether the change in annual incidence of the malformations is associated with changes in the parity composition of the general population of births, we require, of course, the birth rank of malformed individuals and of the related population of births. This evidence is available in Birmingham for the years 1942-49, and Table III gives the incidence of anencephalus and spina bifida in infants in birth ranks 1 to 5* standardized for parity. The sharp fall in the rates is exhibited after standardization, and the change is if anything more regular than that shown in Table I. It is evident that the decrease in incidence is not explained by changes in the parity composition of the general population of births.

TABLE III

INCIDENCE OF ANENCEPHALUS AND SPINA BIFIDA (STILLBIRTHS AND INFANT DEATHS) IN INFANTS OF BIRTH RANKS 1–5 STANDARDIZED FOR CHANGING PARITY DISTRIBUTION OF BIRTHS
Birmingham 1942–49

Year	Estimated‡ Incidence per 1,000 Total Births	
	Anencephalus	Spina Bifida
1942 and 1943	2·3	2·5
1944 and 1945	2·1	2·1
1946 and 1947	1·6	1·8
1948 and 1949	1·4	1·7

‡ The birth rank distribution of the related population of total births is estimated from a random sample of 897 births, for 869 (97 per cent.) of which birth rank was recorded.

The rates examined in Tables I and III were prepared by combining Birmingham stillbirths and infant deaths attributed to the malformations. Unfortunately national rates cannot be given in the same form, and the only useful data available from national sources for consideration of secular changes in incidence of central nervous malformations are:

(*a*) the infant mortality rate attributed to spina bifida† in England and Wales,
(*b*) the stillbirth rate attributed to anencephalus and spina bifida in Scotland.

(*a*) Fig. 3 illustrates the annual infant mortality attributed to spina bifida in England and Wales for the years 1931-49. The rate was fairly constant from 1932 until 1941; it increased in 1942, fell in each subsequent year until 1948, and rose slightly in 1949. The significance of the change is demonstrated in Table IV,

* Births above birth rank 5 were excluded because of small numbers.

† In Birmingham approximately 70 per cent. of combined stillbirths and infant deaths attributed to spina bifida were infant deaths.

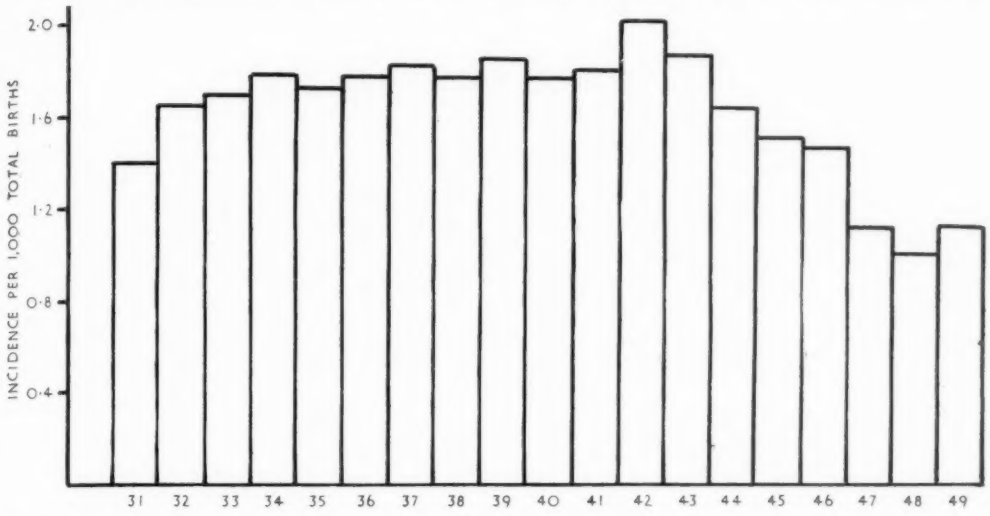

FIGURE 3.—Annual infant mortality rate attributed to spina bifida. England and Wales, 1931–49.

TABLE IV

INFANT MORTALITY RATE (PER THOUSAND LIVE BIRTHS) ATTRIBUTED TO SPINA BIFIDA

Years	England and Wales	Birmingham
1940–1944	1·82	2·03
1945–1949	1·26	1·34
Difference	0·56 ±0·03	0·69 ±0·19

which compares the rates for the two periods 1940-44 and 1945-49. The table also makes the comparison on Birmingham infant deaths which show a similar decrease.

(*b*) Table V compares stillbirth rates attributed to anencephalus and spina bifida in Scotland and Birmingham in the years 1940-44 and 1945-48. In Birmingham, stillborn anencephalics show the decrease already noted in Table I (for stillbirths and infant deaths), but in Scotland there is no significant difference in the rates for the two periods. We are on less firm ground in using the spina bifida

TABLE V

STILLBIRTH RATES (PER THOUSAND TOTAL BIRTHS) ATTRIBUTED TO ANENCEPHALUS AND SPINA BIFIDA

Years	Anencephalus		Spina Bifida	
	Scotland	Birmingham	Scotland	Birmingham
1940–1944	2·57	2·49	0·47	0·87
1945–1948	2·40	1·65	0·39	0·58
Difference	0·17 ±0·11	0·84 ±0·22	0·08 ±0·044	0·29 ±0·13

stillbirth rate as an index of the incidence of spina bifida because of the higher ratio of infant deaths to stillbirths in the case of this malformation. Nevertheless, it seems worth recording that the incidence of stillbirths attributed to spina bifida in Birmingham has decreased since 1940, whereas in Scotland the difference in the rates for the two periods examined is small and of doubtful significance.

Summary

In recent years there has been a considerable reduction in the incidence of some of the more important malformations of the central nervous system as follows:

(*i*) of anencephalus in Birmingham (but not in Scotland),
(*ii*) of spina bifida in Birmingham, England and Wales, and possibly Scotland (although the Scottish data available are inconclusive).

We have no explanation for these secular variations, but are satisfied that they are not associated to any considerable extent with fluctuations in the parity composition of the general population of births. It will be of interest to review these observations in later years, and to extend them to other areas.

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Brit. J. soc. Med. (1951), **5**, 259-264

OBSERVATIONS ON ALL BIRTHS (23,970) IN BIRMINGHAM, 1947

V. BIRTH WEIGHT RELATED TO ECONOMIC CIRCUMSTANCES OF PARENTS

BY

J. R. GIBSON and THOMAS McKEOWN

From the Department of Social Medicine, University of Birmingham

This communication presents evidence on the association of the birth weight of children born in Birmingham during 1947 with the economic circumstances of their parents. This matter was considered by Baird (1945), who recorded differences in the proportion of infants born prematurely (as identified by weight) in nursing home, hospital, and specialist practice, and more recently by Douglas (1950), who noted slight but unimportant weight differences between children of professional and manual workers. Such comparisons clearly require some consideration of the effect of birth rank (as provided by Douglas, who dealt separately with first and later births), since weight is correlated with birth rank and the proportion of individuals in each birth rank is not constant in different economic groups.

As indices of the economic circumstances of the parents we have used a classification of Birmingham wards into three groups of which details were provided by Record and McKeown (1949). Briefly, the city wards were classified using data from:

(1) The Housing Survey Report (1946), which gave for each ward the number of houses:
   - (*a*) back to back
   - (*b*) with bathroom
   - (*c*) with bath
   - (*d*) municipally-owned.

(2) The Birmingham City Statistical Office, which supplied the average rateable value for each ward.

(3) The Town Planning Department, which gave the surface area of each ward, from which were calculated (by wards):
   - (*a*) housing densities
   - (*b*) population densities, using the ward population given in the Report of the Medical Officer of Health, 1947.

This classification is essentially based on the standard of housing, which provides an index of the economic circumstances of residents. As might be

TABLE I

PERCENTAGE DISTRIBUTIONS OF BIRTHS BY WEIGHT

Birth Weight		Ward Group					
		I		II		III	
Pounds	Half-pounds	Number	Per cent.	Number	Per cent.	Number	Per cent.
1	2	1	0·10	3	0·10	—	0·13
	3	7		7		5	
2	4	20	0·54	21	0·45	7	0·46
	5	25		23		11	
3	6	28	0·67	27	0·64	13	0·94
	7	28		36		24	
4	8	73	2·04	69	1·60	24	1·52
	9	97		87		36	
5	10	219	7·43	248	6·60	99	6·48
	11	399		396		157	
6	12	852	24·37	852	21·95	350	22·91
	13	1,174		1,291		555	
7	14	1,557	34·66	1,928	36·30	786	35·89
	15	1,324		1,616		632	
8	16	1,199	21·12	1,483	22·82	602	23·66
	17	557		745		333	
9	18	410	7·00	516	7·37	185	6·07
	19	171		204		55	
10	20	122	1·80	146	1·91	59	1·77
	21	28		41		11	
11	22	12	0·23	18	0·21	6	0·18
	23	7		3		1	
12	24	2	0·02	1	0·02	—	—
	25	—		1		—	
Over 12	Over 25	1	0·01	—	—	1	0·03
Number of Births ..		8,313	100	9,762	100	3,952	100
Mean (lb.)		7·38	—	7·46	—	7·43	—
Error of Mean		0·014	—	0·013	—	0·019	—
Standard Deviation ..		1·28	—	1·25	—	1·22	—

Of 22,454 births (see Table III, Part II), 323 are excluded as previously indicated, and 104 others because the ward group was not known.

expected, the ward groups so identified differ considerably in respect of live birth rates and infant mortality rates, as is indicated by data for 1946-47 which are as follows:

Ward Group	I	II	III	All Wards
Birth Rate	25·7	21·0	19·7	22·4
Infant Mortality Rate	49·2	36·5	33·1	40·4

1947 births were assigned by place of domicile to one of the three groups; Table I gives percentage distributions by weight and the mean weights of births in each group. Of the original 22,527 births of known weight (97 per cent. of all single births in Birmingham during 1947), the following have been excluded:

(*a*) 73 in which the duration of gestation was less than 28 weeks
(*b*) 323 as indicated in Table III, Part II
(*c*) 104 for which ward group was not known.

Mean birth weight is slightly but significantly lower in Group I (in which housing standards are poorest) than in either of the other two groups; the difference in mean weights between Groups II and III is not significant. Weight distributions have also been compared by grouping births by weight in lb. (less than 4; 4—; $5\frac{1}{2}$—; 7—; 8 and over). The χ^2 test gives values for p as follows:

I compared with II and III $p<0{\cdot}0001$
II compared with III $p\simeq 0{\cdot}5$.

We may now inquire whether these results are influenced by the differences in birth-rank constitution of births in the three ward groups exhibited in Table II, in which it is shown that the proportion of births at low birth ranks is highest in wards with better housing. Table III (overleaf) gives mean weights by birth rank, and shows the well-known increase in weight from the first to the third birth. Hence it might be expected that the difference in mean weights already noted in Table I

TABLE II

PERCENTAGE DISTRIBUTION OF BIRTHS BY BIRTH RANK

Birth Rank	Ward Group							
	I		II		III		Total	
	Number	Per cent.	Number	Per cent.	Number	Per cent.	Number	Per cent.
1	2,746	35·59	3,560	40·89	1,615	45·18	7,921	39·61
2	2,217	28·74	2,517	28·91	1,012	28·31	5,746	28·73
3	1,213	15·72	1,209	13·88	509	14·24	2,931	14·66
4 and over	1,539	19·95	1,421	16·32	439	12·23	3,399	17·00
Total ..	7,715	100	8,707	100	3,575	100	19,997	100

Ward group was unknown in 104 of 20,101 births of known birth rank (see Table V, Part II).

TABLE III

MEAN BIRTH WEIGHT RELATED TO BIRTH RANK

Birth Rank	Ward Group			
	I	II	III	Total
1	7·08 ±0·024	7·23 ±0·020	7·20 ±0·031	7·17 ±0·014
2	7·41 ±0·027	7·50 ±0·025	7·52 ±0·038	7·46 ±0·017
3	7·65 ±0·036	7·64 ±0·037	7·71 ±0·053	7·65 ±0·026
4 and over	7·58 ±0·037	7·77 ±0·037	7·61 ±0·070	7·65 ±0·026
All Birth Ranks	7·38 ±0·014	7·46 ±0·013	7·43 ±0·019	7·43 ±0·008

would be increased when births in the same rank are examined. The effect is mainly on first births, for which the means are:

7·08 in Group I
7·23 in Group II
7·20 in Group III

The differences between Groups I and II are also substantial at birth ranks " 4 and over ", but as numbers are smaller the standard errors are considerably greater.

Another question of interest in this context is the relation between duration of gestation and economic circumstances of parents. It is commonly stated that the incidence of " premature " birth is higher among poorer mothers, but the index of maturity in such calculations has invariably been birth weight. Any substantial difference in the distributions by duration of gestation would, of course, influence weight at birth. Table IV gives the distribution of births by duration of gestation for each of the three ward groups. There are no significant differences in the means, which are:

280·76 in Group I
280·73 in Group II
280·58 in Group III

The data suggest that there are no conspicuous differences in the percentage distributions, and when the births are grouped by duration of gestation in weeks (35 and under; 36—; 39—; 42 and over) the χ^2 test gives $p \simeq 0{\cdot}5$. Finally the association between mean weight, duration of gestation, and ward group is considered in Table V. There is here no consistent relationship between weights of births in each group.

TABLE IV

PERCENTAGE DISTRIBUTION OF BIRTHS BY DURATION OF GESTATION

Duration of Gestation (weeks)	Ward Group					
	I		II		III	
	Number	Per cent.	Number	Per cent.	Number	Per cent.
28	5	0·08	8	0·11	3	0·10
29	3	0·05	10	0·14	3	0·10
30	7	0·11	11	0·15	5	0·16
31	23	0·36	14	0·19	13	0·42
32	14	0·22	21	0·29	8	0·26
33	34	0·54	30	0·41	12	0·39
34	44	0·69	43	0·59	24	0·78
35	99	1·56	125	1·72	41	1·34
36	191	3·01	178	2·45	66	2·16
37	290	4·57	332	4·57	137	4·47
38	554	8·73	651	8·96	273	8·91
39	1,266	19·95	1,487	20·46	625	20·41
40	1,792	28·24	2,034	27·99	937	30·60
41	1,142	18·00	1,335	18·37	534	17·44
42	524	8·26	660	9·08	230	7·51
43	212	3·34	188	2·59	94	3·07
44	98	1·54	91	1·25	39	1·27
45	47	0·74	47	0·65	18	0·59
46	1	0·02	4	0·06	—	—
Number of Births ..	6,346	100	7,269	100	3,062	100
Mean (days)	280·76	—	280·73	—	280·58	—
Error of Mean	0·18	—	0·17	—	0·25	—
Standard Deviation ..	14·36	—	14·14	—	14·05	—

Ward group was unknown in 72 of the 16,749 births referred to in Table III, Part II.

TABLE V

BIRTH WEIGHT RELATED TO DURATION OF GESTATION

Duration of Gestation (weeks)	Ward Group					
	I		II		III	
	Number of Births	Mean Weight (lb.)	Number of Births	Mean Weight (lb.)	Number of Births	Mean Weight (lb.)
28–	8	2·00	18	2·33	6	2·33
30–	30	3·07	25	2·96	18	2·83
32–	48	3·79	51	3·65	20	3·65
34–	143	5·34	168	5·37	65	5·49
36–	481	6·25	510	6·18	203	6·18
38–	1,820	6·74	2,138	6·88	898	6·86
40–	2,934	7·22	3,369	7·23	1,471	7·19
42–	736	7·31	848	7·39	324	7·25
44–	146	7·34	142	7·39	57	7·38
Total	6,346	—	7,269	—	3,062	—

SUMMARY

The standard of housing has been used as a basis of classification of Birmingham city wards into three groups, to which single births for the year 1947 have been allocated by place of domicile. It is shown that birth weight is slightly but significantly lower in the poorest group than in the other two groups. This difference is not accounted for by differences in the birth rank of births in each group, and is greater when first births are compared. There are no differences in duration of gestation, as measured by the mean and percentage distribution.

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BOOK REVIEWS

The Social Consequences of Pneumoconiosis among Coalminers in South Wales. By P. HUGH-JONES and C. M. FLETCHER. 1951. Medical Research Council Memorandum No. 25. H.M.S.O., London. Pp. 54. 1*s.* 9*d.*

The main South Wales coalfield extends from Newport in the east to Kidwelly in the west, a distance of some 60 miles. It is bounded on the north by the mountains and on the south by the coastal plain and the Bristol Channel. In the plain lie Cardiff and Swansea, the only two large cities of Wales. Rivers run down from the hills, and in their valleys are the mining villages with good transport north and south, but scanty communication from east to west. The miners and their families make up closed village communities, whose existence depends on coal. For generations mining was the only industry, and a trade depression threw the whole male population out of work. The staple industry is itself particularly dangerous, leading to much illhealth and disability. Therefore, even in times of prosperity, the man who is unfit for mining has difficulty in finding another job, and the absence of light industry prevents the women from going out to work when the need arises. Within the last quarter of a century an effort has been made to introduce other types of industry into South Wales, and factories have been established which offer work not only to women, but also to partially disabled miners. Intensive multilateral research has been conducted into the causes and extent of dust disease of the lungs, and into the control of dust in mines. (There is no doubt that the dust hazard is at present increasing as a result of the introduction of coal-cutting machinery.)

That the social and economic problems of the South Wales coal-miner are far from solved is plain from this report from the Pneumoconiosis Research Unit in Cardiff. Between 1931 and 1948, 19,000 men in this area were certified as suffering from pneumoconiosis. In the same period, in the rest of Great Britain, from a total mining population six times that of Wales, only 3,000 cases were certified. This means that pneumoconiosis is forty times as common in Wales as in the rest of the United Kingdom. There are now about 16,000 certified men alive, of whom 5,000 are unemployed. In addition, there are certainly many more uncertified men, many of them also out of work. In October, 1948, 2 per cent. of the insured male working population in Great Britain was unemployed, but in S. Wales the proportion was 5 per cent. Registered disabled persons amounted to 7·3 per cent. in Gt. Britain and 21·6 per cent. in S. Wales, and of the men disabled by pneumoconiosis in S. Wales, 5,000 (40·3 per cent.) were out of work. The authors consider that three-quarters of these could work under normal industrial conditions.

Pneumoconiosis usually takes many years to reach the stage when symptoms impel a man to apply for certification. From 645 applicants in 1938, the numbers have risen yearly, reaching a peak in 1945, when 9,117 men applied, 57 per cent. of them being granted certificates of disablement. By 1947 the applications had dropped to 7,748, of whom only 36 per cent. were certified.

Fletcher has suggested that the great increase in certification during the war years was due to several factors: the introduction in 1943 of the Pneumoconiosis Scheme, under which many men formerly outside the existing compensation schemes could claim benefit; the increasing awareness among miners that chest symptoms might be the result of pneumoconiosis and therefore compensatable; the Essential Work Order under which the easiest way to obtain release from work was on medical grounds; and finally, increased dust concentration from mechanization of mining processes, leading to a real increase in the number of cases.

Until July, 1948, when the National Insurance (Industrial Injuries) Act replaced the old compensation laws, certification carried with it the further disability of suspension from mining. This unfortunate policy led to much bitterness through enforced unemployment and undoubtedly reduced the numbers who might have applied for benefit. Now, certain men are allowed to continue working under " approved conditions ", provided they are not also suffering from superimposed tuberculosis.

The South Wales problem is beset with every conceivable adverse factor. The nature of the disease itself presents difficulties. Correlation between radiological appearances and degree of disability is low, and prognosis is uncertain—some cases progress steadily, others remain stationary or may even regress. Because the chief symptom is breathlessness on exertion, many men are unable to manage the long hilly walks which normally separate their homes from the buses running up and down the valleys. If they can surmount this obstacle, the only work obtainable is often heavy, unskilled labouring—quite unsuited to their disability. If they can manage the work, the job, being casual, frequently ends in redundancy. The available light work is often taken up by women, and many employers are reluctant to take on men disabled through pneumoconiosis.

The medical aspects of the disease, including the grading system used in the Cardiff Unit, are discussed and the legal aspects surveyed. The report is concise, well-documented, and informative. It poses many questions, few of them purely medical, the most important problem being the control of dust in the mines without which pneumoconiosis is inevitable. To rehabilitate those already disabled, a far greater national effort is required to establish alternative industries in the mining valleys, for the disabled men must have a chance to be useful members of the community, able and willing to do a reasonable day's work.

This report should be read by all concerned with the social and economic problems of Great Britain as well as by workers in the field of industrial medicine.

CATHERINE SWANSTON

New Discoveries in Medicine: their Effect on the Public Health. By PAUL R. HAWLEY, M.D. 1950. Columbia University Press, New York. Pp. 134, 15 illus. ($2.50; 16*s.*).

In the Bampton lectures delivered at Columbia University in 1949, Dr. Paul Hawley reviews some of the more dramatic advances made in medical science during the past quarter of a century. His first talks, devoted to what he appropriately calls " our fabulous blood ", include an excellent account of the practice of blood transfusion and of the advances made in the two world wars. He gives a clear picture of the mechanism of the Rh factor, but his suggestion that the young man who is an Rh-positive heterozygote should be told this at an early age and advised to " avoid mating with an Rh-negative female " is unlikely to be taken seriously. It is to be feared that love laughs not only at locksmiths but also at biological obstacles.

In other lectures Dr. Hawley describes some modern developments in surgery, especially in thoracic and mental disorders, the latter with particular reference to pre-frontal leucotomy. Finally he discusses the organization of medical services in a community, and briefly describes the various insurance schemes operating in the United States for the prepayment of medical costs. He reviews the National Health Service in Great Britain, and, though endorsing the principle, points out the inevitable early defects of its implementation, showing that increased patient-demands with no corresponding increase in medical personnel have led to more medical attention but less personal medical care. Abuses of the service are impossible to prevent, and its costs have been much underestimated. There is much truth in these criticisms, as three years of nationalized medicine have all too clearly shown, but neither Dr. Hawley nor anyone else seems able, as yet, to suggest a remedy.

This is a readable little book, presenting useful information in an attractive manner.

CATHERINE SWANSTON

ABSTRACTS

(This section of the JOURNAL *is devoted to selected abstracts of articles on social medicine appearing in the current literature. The section will be edited in collaboration with the two abstracting Journals,* ABSTRACTS OF WORLD MEDICINE, *and* ABSTRACTS OF WORLD SURGERY, OBSTETRICS, AND GYNAECOLOGY.)

Report on Intestinal Parasitism in Children of School Age. (Rapport sur le parasitisme intestinal chez les enfants d'age scolaire.) COUTELEN, F. (1950). *Gaz. méd. France*, **57**, 857. Bibl.

This paper from the Medical School at Lille gives the results of stool and adhesive-cellulose-tape perianal examinations of 6,202 school-children in Northern France. The technique of diagnosis of *Oxyuris* infection by means of " cellophane " tape was that introduced by Graham (*Amer. J. trop. Med.*, 1941, **21**, 151). After a discussion of the method of examination employed, the results are presented in two groups as follows: (1) children 2 to 6 years of age at nursery schools; (2) children in older age groups temporarily resident in institutions.

1. (*a*) Of 1,586 children 2 to 6 years old at nursery schools (4,384 stools examined), 68·64 per cent. were found to be infected. Of these, 27·77 per cent. had *Entamoeba coli* and 28·86 per cent. *Giardia intestinalis*, and small numbers were infected with seven other protozoa; none had *Entamoeba dysenteriae*. Worms or ova of *Oxyuris* were present in the stools of 21·83 per cent., of *Ascaris* in 7·15 per cent., and of *Taenia saginata* in 1·32 per cent.

(*b*) Of 332 nursery-school children from a crowded quarter of Lille examined by three stool and three cellulose-tape tests, 96·38 per cent. had oxyuriasis.

(*c*) Of 3,440 nursery-school children in Lille subjected to one stool examination and one adhesive-tape test, 59·3 per cent. were shown to be infected, including 25·7 per cent. with *Giardia* and 39 per cent. with *Oxyuris*.

2. (*a*) Of 250 children aged 1 to 15 years in the surgical clinic at Lille, 88 per cent. were infected: 20·03 per cent. with *Ascaris*, 42·9 per cent. with *Trichocephalus*, and 64·5 per cent. with *Oxyuris*. Of 49 girls in the last group in whom positive results of the Graham test for oxyuriasis were found, in 77·5 per cent. vulval swabs were also positive.

(*b*) Of 160 children aged 2 to 15 years in the medical clinic at Lille, 60·63 per cent. were infested with alimentary parasites (including *Oxyuris*). Of 41 girls in this group positive results of the anal test for *Oxyuris*, in 37 positive vulval swabs were also found.

(*c*) Of 100 children aged 1 to 19 years in a sanatorium-preventorium, 98 per cent. were infected. The highest percentage of giardiasis occurred among the 2-to-4-year age group; among the older children 44 per cent. had ascariasis, 80 per cent. trichocephaliasis, and 75 per cent. oxyuriasis.

(*d*) Of 334 children aged 6 to 14 years in a psychiatric institution, 91·31 per cent. had oxyuriasis.

The most striking features of the investigation were the general absence of *Entamoeba dysenteriae* and the high incidence of ascariasis among older children in the sanatorium.

There is a valuable list of 177 references to surveys of alimentary infestation carried out in 43 countries.

Ronald MacKeith

Acute Appendicitis in Children. A Review of One Thousand One Hundred and Sixty-five Cases. ABEL, W. G., and ALLEN, P. D. (1950). *Ann. Surg.*, **132**, 1093. 4 figs, 31 refs.

This is a review of the results of operation for acute appendicitis performed in connexion with the Children's Surgical Service, Bellevue Hospital, during the period 1926 to 1947 inclusive. All the children were under 12 years of age. The total number was 1,165. During the period under review there has been a gradual lessening of the number of cases admitted; one reason adduced is the greater number who prefer private treatment.

The cases are classified as those with unperforated appendix, with abscess, and with spreading peritonitis. The number of cases with abscess has steadily diminished from 25 to 7·6 per cent. of the total, while the number of cases in which the appendix was unperforated has increased from 51 to 72·6 per cent. There has been only a slight decline in the percentage of cases with spreading peritonitis.

The total mortality has decreased from 5·8 to 1·1 per cent., with an average for the whole period of 3·8 per cent. From 1941 to 1947 there were no deaths among those with unperforated appendix or with abscess. The mortality for those with spreading peritonitis during the same period was 5·8 per cent. Among the factors in the great improvement are said to be the following: the patients came to the surgeon earlier, there was less catharsis; the electrolyte balance was corrected; the Levin tube was available; the McBurney incision was more regularly used; and better antibacterial agents were available.

In spite of the excellent results published by Johnston and Ochsner in the conservative treatment of spreading peritonitis, the authors consider that penicillin should remain as an adjunct only to competent surgery.

Zachary Cope

A Further Study of the Teeth of 5-year-old Children in Residential Homes and Day Schools. MELLANBY, M., and MELLANBY, H. (1951). *Brit. med. J.*, **1**, 51. 17 refs.

In continuation of an extensive study which has been in progress since 1929, this paper records the results of the examination, in 1950, of the teeth of 542 children aged 5 years in residential institutions, as compared with 560 children of the same age attending private day schools in or near London. The findings in these two groups are compared with each other and with those in similar groups examined in 1945 and 1949. The institutionalized children had, on the average, poorer tooth structure than the private school children, though the difference was less marked than it had been in former years. The incidence of caries was 7·2 per cent. in the institutionalized children (10·5 per cent. in 1945) and 12·7 per cent. in the private school children (14·2 per cent. in 1945). The conclusion was reached that the institutionalized children received a better diet, which more than made up for the increased susceptibility to caries to which they were prone from their poorer tooth structure. A high proportion (33 per cent.) of institutionalized children showed brown or black staining of the teeth, but this did not seem to be related to susceptibility to caries.

D. Gairdner

Sleep Problems in the First Three Years. ILLINGWORTH, R. S. (1951). *Brit. med. J.*, **1**, 722. 6 refs.

After commenting on the paucity of literature on this subject, the author gives an outline of his own conclusions and experience. He stresses first the necessity of understanding the normal. After 4 months of age the child acquires sleep habits which may, at 2 or 3 years, develop into a more and more complicated ritual if unchecked. The great variation in the amount of sleep required by different children, and by the same child at different times, is emphasized. The importance of accepting the difficult phase of ego development between 9 months and 3 years is stressed.

After outlining what may be described as the normal routine, which should remain free from regimentation, the possible difficulties are described under four headings: bed refusal, sleep refusal, night waking, and early morning waking. The principal factors in the approach to the problem are love and understanding. Firmness must be combined with patience, and a rigid regimen is decried. Drugs are only to be used for a particular purpose such as breaking a difficult habit due to mismanagement, and only as a last resort. The possible need for treating the mother in order to help the child must be remembered. Each case is an individual problem.

E. H. Johnson

Intussusception in Children under Two Years of Age. An Analysis of Fifty-four Cases from Charity Hospital of Louisiana at New Orleans. KAHLE, H. R. (1951). *Surgery*, **29**, 182. 9 refs.

An analysis is given of 54 cases of intussusception admitted to the Charity Hospital, New Orleans, from 1938 to 1949. The mortality of 25·9 per cent. compares favourably with that of 63·6 per cent. for a reported series over the previous 34 years, but is still far too high. The sex ratio was two males to one female, and a study of ages revealed fifty cases in the first year of life, of which 41 occurred between 3 and 8 months. The study revealed no obvious aetiological factor. The symptoms followed the classical pattern, with pain occurring in all, vomiting in 43, and bloody stools in 49 patients. An abdominal mass was palpable in 32 patients and a lump per rectum in seventeen. *X*-ray examination of the abdomen was sometimes helpful, but in most cases not really necessary.

Reduction occurred spontaneously in five children. In five other cases a barium enema reduced the intussusception, but the condition is still regarded as a surgical emergency in spite of the favourable results of hydrostatic-pressure reduction reported elsewhere. One risk of this method is the possibility of missing gangrenous bowel. Also, in most successful series of reduction by non-surgical methods the time lapse between the onset of symptoms and the institution of therapy was very much lower than in most surgical series. However, the method is now being given a trial.

Of the 54 cases, fifty came to operation: thirty patients had a simple reduction of the intussusception, and seven of these died. In a further ten cases treatment was by reduction combined with a further procedure such as appendicectomy or suture of a tear; four of these patients died. In four cases a resection was necessary and three of these children died. Undoubtedly the chief factor in mortality was the delay in admission; next came poor judgement at operation (such as replacing non-viable gut), rupture of the bowel during reduction, too much surgery, and poor post-operative resuscitation.

Intestinal obstruction from post-operative adhesions occurred in four cases after intervals of 5 weeks to 4 years: two of these children died. To avoid this serious complication it is suggested that patients in whom there is evidence of post-operative infection should receive chemotherapy.

A. J. Drew

Critical Evaluation of Antihistaminic Drugs in the Common Cold. FABRICANT, N. D. (1950). *Arch. Otolaryng., Chicago*, **52**, 888. 22 refs.

" As the result of extravagant and irresponsible claims in behalf of antihistamine drugs, the American public has been deluded into spending huge sums of money on the ' sensational new discovery that kills colds in hours '. It will continue to do so until it is educated by alert physicians to the foolishness of such practice." The author amplifies these remarks with a list of dangerous complications produced by the antihistamine drugs in patients taking them for the common cold: three cases of haemolytic anaemia, three of agranulocytosis, rashes, cardiac excitation, headache, tinnitus, fever, as well as the better known effects such as drowsiness and dizziness. In a survey of 247 physicians 223 reported having noted side-effects of antihistamine drugs in their practices.

There is no real evidence that allergy is mainly responsible for the common cold. Most of the writers who advocated these drugs for the common cold failed to differentiate between the infective cold and vasomotor

disturbances or allergic conditions. Fully controlled studies in the United States and in Great Britain have demonstrated the failure of antihistaminics in the treatment and control of the genuine "common cold". To these studies is now added a carefully controlled research on 213 students. It is concluded that "antihistaminic drugs do not prevent, abort, shorten, curtail, reduce, or stop the common cold. It was observed that antihistaminic drugs are no more effective than placebos in aborting a cold and that there is no validity to the contention that antihistaminic drugs are more effective if taken within a short time after the start of a cold".

F. W. Watkyn-Thomas

Q Fever Studies in Southern California. Summary of Current Results and a Discussion of Possible Control Measures. HUEBNER, R. J., and BELL, J. A. (1951). *J. Amer. med. Ass.*, **145,** 301. 4 figs, 24 refs.

This is a summary of the work done on Q fever in the Los Angeles area since 1947, when it was first recognized there. Its discovery was followed by studies on cattle and milk, a survey of 300 clinical cases, and epidemiological studies embracing almost 10,000 persons. Antibodies against Q fever were demonstrated in thirty widely separated herds and in the pooled milk of many dairies: 45 per cent. of those affected resided within a quarter-mile (0·4 km.) of a dairy, the occupation of 38 per cent. brought them into contact with livestock, and 32 per cent. had used raw milk in their households. Agglutination tests in various occupational groups gave 23 per cent. positive results in the dairy trade, 21·6 per cent. in fat-rendering plants, and 16 per cent. in hide and wool plants, compared with 1·4 per cent. in the aircraft industry. Pasteurization of all milk supplies and vaccination of exposed persons are immediately practicable measures, but effective defence depends on the control of animal reservoirs of infection.

W. G. Harding

Observations on the Epidemiology of Q Fever in Northern California. LENNETTE, E. H., and CLARK, W. H. (1951). *J. Amer. med. Ass.*, **145,** 306. 12 refs.

Outbreaks of Q fever in California have been associated with a high percentage of positive agglutination reactions in sheep, goats, and cattle herds respectively. It is suggested that animal excreta and secretions infected with rickettsiae may be potent sources of infection, possibly by dust spread followed by ingestion or inhalation.

W. G. Harding

Typhoid and Paratyphoid Endoanatoxins in the Prophylaxis of Enteric Fevers. Application and Collected Results of 15 Years' Immunization (1934-1948). (L'endoanatoxine typhoparatyphique dans la prophylaxie des infections typhoïdiques. Applications et résultats d'ensemble de quinze ans de vaccination (1934-1948). GRASSET, E. (1951). *Rev. Immunol.*, **15,** 1.

This is a review of 15 years' experience, mainly in South Africa, with typhoid endotoxoid vaccine in the prevention of enteric fever. Prepared according to the technique of the author, it is considered to be a highly potent immunizing agent, while less prone than classical T.A.B. vaccine to cause undesirable after-effects.

After a preliminary controlled trial in which it appeared to be the method of choice it was successfully used on a large scale to immunize native mine-workers on the Rand—an unstable population living in poor sanitary conditions with enteric fever always present. Following this, its use was extended to other industrial workers, to those engaged in major public works such as irrigation schemes, and to civilians generally. On the outbreak of war in 1939 it was introduced into the Services and later was used for immunizing prisoners of war and civilian internees.

The decreased incidence of enteric fever reported, whether resulting wholly or partly from the use of this vaccine, presents a dramatic picture.

A. D. Macrae

A Study of Reactions following Administration of Crude and Purified Diphtheria Toxoid in an Adult Population. PAPPENHEIMER, A. M., EDSALL, G., LAWRENCE, H. S., and BANTON, H. J. (1950). *Amer. J. Hyg.*, **52,** 353. 1 fig., 22 refs.

In view of recent work which has shown that there has been an increase in cases of adult diphtheria since the war and that the proportion of Schick-negative adults may be as low as 15 per cent., a study was made of the Schick status of approximately 3,000 U.S. Army personnel. At the same time observations were made on the local and systemic reactions produced by toxoids of varying degrees of purity. The men were divided into seven groups of 400, each group receiving a different batch of toxoid and all receiving the Schick test (0·08 units of crude toxoid) and Schick control. All toxoids were diluted to contain ten flocculating units per ml.; the Schick-test material contained 1/50 MLD in 0·1 ml. Crude toxoid, toxoid partially purified by ammonium sulphate fractionation, and toxoid highly purified by alcohol fractionation were used, the purity of the three types of preparation being 20 per cent., 70 per cent. and 95 per cent. respectively on the basis of protein nitrogen content.

The first examinations were carried out 18 to 20 hours after inoculation. Men showing local or systemic reactions (approximately 10 to 20 per cent. of all men in any given group) were again examined 24 hours later. Of all men 40 per cent. were Schick positive, the highest proportion (50 per cent. being in the under-20 age group and the lowest (36 per cent.) in the over-35 age group. The frequency of allergic reactions to toxoid increased in the older age groups. With crude toxoid, the incidence of local reactions remained almost constant in all age groups, but partially purified toxoid gave significantly fewer reactions in the younger groups as compared with the older groups. The same gradation was seen with the purest toxoid and in the frequency of systemic reactions.

As would be expected, there were few local or systemic reactions in Schick-positive subjects. Injection of highly purified toxoid produced significantly fewer and less severe reactions than those obtained with all equivalent dose of crude toxoid in Schick-negative subjects. Almost

all individuals showing local or systemic reactions gave an allergic response to the Schick control inoculation, irrespective of the result of the Schick test itself; men showing a " two plus " reaction to the control test almost invariably had a marked systemic reaction to crude toxoid, and approximately 50 per cent. reacted similarly to purified toxoid.

In confirmation of the work of other workers it was found that there is an inverse correlation between the titre of serum antibody and the flocculation time of the toxoid. *J. F. McCrea*

Poliomyelitis and Diphtheria Immunization in Belfast. McLeod, W. J. (1951). *Brit. med. J.*, **1**, 736. 5 refs.

The experience of Belfast on the relation of diphtheria immunization to the poliomyelitis epidemic of 1950 is discussed. A total of 98 cases of poliomyelitis were reported up to September 19, of which ten were non-paralytic. The incidence under 5 years was thirteen per 10,000 of the population in this age group. The epidemic wave gradually rose from the middle of March to a peak in the middle of July. Immunization against diphtheria continued as usual until the holiday closure of the clinics on July 8. Administrative action was taken in Northern Ireland late in July to discourage immunization during the epidemic. It would seem, (although not precisely stated) that combined diphtheria-pertussis inoculation was almost entirely suspended during the epidemic. It is estimated that some 2,000 inoculations of A.P.T. were given by general practitioners during the epidemic, (presumably nearly all before the end of July, that is, during the first half of the epidemic). In this group one " double event " occurred—inoculation into the right hip followed 8 days later by paralysis of the right leg. In addition there was one case of facial paralysis following 23 days after P.T.A.P. inoculation in the left arm.

The suggestion is made that subcutaneous P.T.A.P. inoculation, which produces less local reaction than A.P.T., is a relatively innocuous antigen so far as induction of poliomyelitis in the inoculated limb is concerned. This is based on the complete absence of association between the site of inoculation and the site of paralysis after 6,250 inoculations of P.T.A.P. performed in the clinics " during the epidemic ". [From the text it would seem, however, that these inoculations were given during the period January 1 to July 8. From January to the middle of April there were very few cases of poliomyelitis, and from the middle of April until July 8 presumably no more than one-half of the total number. The negative experience of " double events " with P.T.A.P. is therefore not so favourable as it is represented to be in the discussion and summary of this paper. In this respect the article seems to the abstracter to be somewhat misleading.] *H. Stanley Banks*

Acute Anterior Poliomyelitis in the Seine Department of France, 1935-1950. Epidemiological Deductions. (La poliomyélite antérieure aiguë dans le département de la Seine de 1935-1950. Deductions epidémiologiques.) Boyer, J., Corre, L., Langlois, J., and Tissier, M. (1951). *Pr. méd.*, **69**, 484. 9 figs, 2 refs.

A number of graphs present the relevant data upon which the authors base their deductions on the epidemiology of poliomyelitis in the Seine Department of France between 1935 and 1950. There appeared to be no evidence that movements of civilians and soldiers during the war had influenced the incidence of the disease. Seasonal changes such as coldness of the winter or dryness of the summer likewise showed no obvious relationship. On comparing the incidence in the Seine district and in France as a whole it appeared that the incidence in winter was higher in towns than in the country districts (but the authors bring forward a number of trivial reasons to avoid accepting this as evidence favouring a spread by way of the respiratory tract). The graph showing distribution of cases by age and sex is interesting as it points to a declining incidence with rising age and to the fact that males are afflicted more often than females, especially among children under 5 years.

Scott Thomson

Poliomyelitis in Semi-closed Communities. Gale, A. H. (1951). *Lancet*, **1**, 735. 5 figs, 29 refs.

The author summarizes a number of epidemics of poliomyelitis occurring in closed communities between 1917 and 1949. These included four outbreaks in school camps, four in children's homes, the Broadstairs epidemic of 1926, and 31 outbreaks in boarding schools in England and the U.S. The author points out how difficult it is to trace any clear relationship between seasonal incidence, severity, and duration of an outbreak with the type of community involved. In camp outbreaks there seems to be a high incidence of paralytic cases; in children's homes a notable feature has been the high attack rate among staff. In schools there is at present no means of forecasting what is likely to happen when a paralytic case occurs, the difficulty being that in the early stages dissemination of the virus is at first a silent process. Multiple paralytic cases have rarely been encountered in schools for girls, and it is suggested that this may be due to the fact that girls are more likely to be confined to bed for comparatively trivial ailments than are boys. Outbreaks are certainly commonest in the autumn term, less so in the summer term, and very rare in the spring term, though they may occur at any period within a given term. The author stresses difficulties in the epidemiological study of poliomyelitis and points out the value of uniform methods of presenting clinical accounts of an outbreak. No new suggestion is made as to the best method of dealing with a localized outbreak, but once again the evil effects of exercise in the early stages of the disease are emphasized. In general the advice tendered by the Medical Officers of Schools Association in 1946 is regarded as the soundest at present available.

Joseph Ellison

A Milkborne Poliomyelitis Episode. Lipari, M. (1951). *N.Y. St. J. Med.*, **51**, 362. 4 figs, 4 refs.

The author describes an epidemic of poliomyelitis which occurred in Delaware County, N.Y., during late October, 1949. At this time poliomyelitis was not prevalent in the district, ten cases only having been reported during the summer, one from the area under

review. From September 26 to November 20 a total of 23 cases were reported from a locality with a population of 3,000 persons. During the same period nine cases occurred in the rest of the county. Much evidence is collected to show that the outbreak was disseminated from a single dairy where the sole operator was the father of one of the patients. It was not, however, possible to prove that infected milk was the sole cause of spread in this epidemic, though it seems clear that its importance was paramount. Exceptionally high temperatures for the time of year are regarded as one important factor in fomenting this outbreak.

Joseph Ellison

Age Incidence and Seasonal Development of Neutralizing Antibodies to Lansing Poliomyelitis Virus. TURNER, T. B., HOLLANDER, D. H., BUCKLEY, S., KOKKO, U. P., and WINSOR, C. P. (1950). *Amer. J. Hyg.*, **52,** 323. 8 figs, 32 refs.

The authors have carried out an extremely comprehensive study of the epidemiology of poliomyelitis in Baltimore City. Among the questions investigated were: (1) the total incidence of infection; (2) the level of infection in different age groups; (3) the seasonal variations in infection; (4) the proportion of infections associated with clinical manifestations.

Clinical and serological findings were obtained from 970 persons, the majority being children less than 15 years old, who were admitted to the Johns Hopkins Hospital, Baltimore, between the years 1941 and 1948. Physical examination was carried out on each patient, and serum samples taken at intervals. In the routine neutralization test equal volumes of serum were stood for one hour with 200 LD50 of Lansing poliomyelitis virus; eight mice were inoculated with each mixture and the results recorded as positive if more than half survived the 21-day period of test.

Neutralizing antibodies were present in 72 per cent. of sera from infants less than 3 months old. The number of positive sera decreased rapidly thereafter until a figure of approximately 10 per cent. was reached at the end of the first year. The proportion then increased to 50 per cent. at 4 years, 72 per cent. at 5 to 9 years, 84 per cent. at 10 to 14 years, and 90 per cent. at 15 years and over. There was no significant variation with sex, but negroes showed a higher infection rate than whites. In no case did the serum test revert from positive to negative during the 7-year period of the test, but the data do not indicate whether this is due to the persistence of antibody or to repeated " booster " infections. Approximately 20 per cent. of previously negative children become positive each year. Infection was largely confined to the summer months, reaching a peak during August and September as judged both by notification of cases and appearance of antibodies.

A mathematical model was developed to express infection rates. Families of curves were constructed taking into account the dynamic equilibrium existing between immune and non-immune sections of the population. On this basis theoretical attack rates and age-distribution curves were calculated and found to agree within experimental error with those obtained from observational data. An estimate of mean annual attack rate at any age level can also be made from such curves.

J. F. McCrea

The Inheritance of the Sickling Phenomenon, with Particular Reference to Sickle Cell Disease. NEEL, J. V. (1951). *Blood*, **6,** 389. 5 figs, 25 refs.

The sickling phenomenon in erythrocytes has been studied in 465 individuals from 75 families. They were all negroes from the south-east of Michigan, and the families were selected by the presence in each of at least one member with either the sickle-cell trait or sickle-cell anaemia. Each individual was classified according to the finding of sickle-cell anaemia, the sickle-cell trait, or an entire absence of sickling. The decision as to whether an individual exhibited sickle-cell anaemia or the sickle-cell trait was made on the following criteria: (1) the presence of anaemia; (2) the presence of evidence of increased erythrocyte destruction (elevated serum bilirubin, elevated reticulocyte count); (3) the presence of leucocytosis; (4) the type of sickling observed microscopically; the sickling in sickle-cell anaemia was found to be more elongate and filamentous than in the sickle-cell trait; (5) the clinical history and physical findings. Using these criteria the author had little difficulty in classification. The only confusion arose with infants and young children with sickle-cell trait and a superimposed secondary anaemia in the phase of active recovery with moderate reticulocytosis. In such instances observation of the patient over a period eventually clarified the position.

The familial distribution observed was most readily interpreted on the hypothesis that the sickling phenomenon is due to a gene which in single dose (heterozygous condition) produces only the sickle-cell trait, and in double dose (homozygous condition) produces sickle-cell anaemia. On this hypothesis both parents of each patient with sickle-cell anaemia should show the sickle-cell trait. In fact out of 94 parents of such children there was only one exception to this rule. In the sibships segregating for sickle-cell anaemia the ratio of members with the sickle-cell trait to the normal was approximately 2 : 1, as would be expected on this hypothesis. However, in the sibships resulting from the marriage of a normal person with an individual with the sickle-cell trait, the ratio of sickle-cell trait individuals to normal ones was significantly less than that (1 : 1) expected theoretically.

Harry Harris

The Genetics of Mediterranean Anaemia. (Sulla genetica di alcune emopatie). SILVESTRONI, E., and BIANCO, I. (1951). *Policlinico* (*prat.*), **58,** 673. 4 figs, 22 refs.

The authors describe their own observations on the genetics of Mediterranean anaemia, sickle-cell anaemia, and constitutional elliptocytosis. In 63 families in which one parent had the Mediterranean trait and the other was normal 182 children were born, of whom 91 had the Mediterranean trait and 91 were normal. In seventeen families in which both parents had the Mediterranean trait 82 children were born, and 22

developed Cooley's disease. In a further 51 families in which the propositus was a child suffering from Cooley's disease both parents had the Mediterranean trait, and of 205 children 87 had developed Cooley's disease. These data agree very closely with the hypothesis that an individual heterozygous for the Mediterranean trait shows target cells but is not otherwise affected, whereas a homozygote develops Cooley's disease. Constitutional elliptocytosis has been observed in fourteen individuals, eleven in four families and three isolated cases. Of these, eleven are healthy and three have slight anaemia. It is concluded that elliptocytosis behaves as a Mendelian dominant character.

Reference to a probable homozygote described in the American literature is made. Further, two families showing the sickle-cell trait and two families with sickle-cell anaemia, and a third family with anaemia have been studied partly in Italy and partly by Guyton and Heinley in America. The results show that the (heterozygotic) sickle-cell trait is compatible with robust health, but that the homozygote suffers from severe anaemia. In seven families, one parent had the sickle-cell trait and the other the Mediterranean trait. In one family the father had both Mediterranean and sickle-cell traits and the mother the Mediterranean trait, and in three other families one parent had the sickle-cell trait and the other parent was dead, but the relatives showed the Mediterranean trait. In this group of families approximately one-quarter of the children showed both sickle-cell and Mediterranean traits. These three congenital haematological anomalies are inherited as single factors, but only the homozygote appears to be seriously affected. The double heterozygote, sickle-cell plus Mediterranean traits, is moderately severely affected. Some evidence is available which suggests that if to the genetic background of Cooley's disease is added one factor for sickling, then the disease is more serious.

[This paper includes much material which has been published elsewhere, but it is the most lucid account of these anomalies which has yet appeared in print. It shows quite clearly that the genes for sickle-cell traits and for Mediterranean traits are carried on two different chromosomes and that in the absence of malaria and intercurrent diseases only the homozygote or the double heterozygote suffers because he carries these characters. It is a paper which should be read by all those interested in these congenital anomalies of the erythrocyte. The data do not agree altogether with the results of Lehmann and others on the sickle-cell trait in pygmies of Uganda, where the trait is present in about half the entire population but sickle-cell disease is rare, whereas it should occur in 25 per cent. of the children in such a population.]

G. Discombe

Familial Leukemia. A Report of Leukemia in Five Siblings, with a Brief Review of the Genetic Aspects of this Disease. ANDERSON, R. C. (1951). *Amer. J. Dis. Child.*, **81**, 313. 1 fig., 11 refs.

The author describes a family in which five children of one generation were affected by leukaemia. Brief clinical summaries are included, as well as a reference to similar cases in the literature. One child was observed for 2 years before the typical findings were demonstrated in the blood, another for over 1 year; three unaffected siblings were observed; one showed a leukaemoid picture in the blood, associated with a respiratory infection, which was confirmed by examination of the bone marrow. Necropsy following another respiratory infection did not show evidence of leukaemia. The blood examinations of the parents and the other two siblings were normal. Questioning of 135 members of the family revealed only one case of cancer, and none of blood disorder.

H. G. Farquhar

Blood-groups of Mental Defectives and their Maternal Parents. PANTIN, A. M. (1951). *Nature, Lond.*, **167**, 76. 8 refs.

A random sample of 370 mental defectives in East Anglia was studied: of these, 252 were considered to be " undifferentiated " in that no obvious cause for the deficiency was found. The blood groups of these 252 and their mothers were determined using anti-A, B, C, D, E, and c sera. Of the total, 44·8 per cent. were group O, 46·4 per cent. group A, 6·0 per cent. group B, and 2·5 per cent. group AB. This confirms Race's findings of a slight excess of group A over group O in East Anglia. In 55 cases mother and child were ABO-incompatible: 191 children were D-positive and 61 dd. This is considered to be a statistically significant high figure for dd: 195 mothers were D-positive and 57 dd. In only 24 cases was the child D-positive and the mother dd. These figures do not suggest that ABO- or rhesus-incompatibility between mother and child is a significant cause of mental deficiency.

G. Jacob

Observations on the Frequency of Blood Groups A, B, O, and Rh in Greece. (Quelques observations sur la fréquence des groupes A, B, O ey Rh en Grèce). PANGALOS, G. E., PAVLATOU, M., and ROUSSOU, M. (1951). *Sang*, **22**, 153.

The rarity of Rh-negative blood in Greece (9·9 per cent.) is attributed to the admixture of oriental races, among which Rh negativity is very rare.

A. Piney

A Study of the Natural History of Acute Leukemia with Special Reference to the Duration of the Disease and the Occurrence of Remissions. SOUTHAM, C. M., CRAVER, L. F., DARGEON, H. W., and BURCHENAL, J. H. (1951). *Cancer*, **4**, 39. 6 figs, bibl.

This paper gives details of 38 cases of remission of acute leukaemia recorded in the literature, and clinical and haematological details of 173 cases of acute leukaemia seen at the Memorial Centre for Cancer and Allied Diseases in New York from 1926 to 1948 inclusive. Of these 173 patients, 23 had received folic-acid antagonists and are excluded from the survival and remission figures. The difficulty of classifying some cases is emphasized; for example, selection of cases in which

there are " blast " cells in blood or bone marrow misses some that have not this feature but run an acute course; in some " blastic " cases, which are called " subacute ", the patient has survived more than a year. On the other hand, collection of all cases in which the patient survived less than a year includes some that were clearly in the final stages of chronic leukaemia. The authors therefore took into consideration all cases diagnosed as of acute or subacute leukaemia, all cases of leukaemia in patients under 21 years of age, and all cases in which the duration from the first symptoms to death was 52 weeks or less; they eliminated eleven adults who lived less than 52 weeks because they obviously had chronic leukaemia; and they excluded one case of typical chronic myeloid leukaemia in a child aged 15 and one of eosinophil leukaemia in a child aged 9.

The analyses show that 36 cases were unclassified and the remainder were equally distributed between lymphatic and myeloid types of disease. Most cases occurred between birth and 4 years of age, then the incidence falls to a minimum between 30 and 34, followed by a rise to a still relatively low level at 45 to 49, and again a small peak at 70 to 74 years. Males were more affected than females except in the 4th year of life; sex, on the whole, made little difference to survival. Figures show that 25 per cent. of the patients were dead by the 9th week after symptoms started, 33 per cent. by the 12th week, 50 per cent. in 17 weeks, 66 per cent. in 22 weeks, 75 per cent. in 28 weeks, and 95 per cent. in 40 weeks; only four patients (2·7 per cent.) survived for 1 year. Sex had no influence on survival, and the various cytological types also show much the same survival rates. Treatment, judged by 5-year periods, has increased survival: in 1931-35 the mean survival was 15·4 weeks; in 1941-45, 19·4 weeks; and in the 3 years 1946-48, 23·0 weeks; 1926-30 was exceptional, showing 25 weeks. The methods of treatment used were mostly supportive—that is, blood transfusion and antibiotics; some of the later cases had received *x*-ray therapy, nitrogen mustards, or radio-phosphorus. The leucocyte count when the patient was first seen gives some clue to survival, and, in general, the higher the initial count, the shorter the expected survival; for example, the mean survival of the 25 patients who had less than 5,000 leucocytes per c.mm. when first seen was 25·0 weeks; the survival of those thirty patients with over 100,000 cells per c.mm. was 16·3 weeks.

Brief details of the recorded remissions in the literature are given and they show an extraordinary variation of survival, varying from 7 to 210 weeks, and the 210-week survivor was still alive when last seen. Of such cases 38 are mentioned; most of them had only supportive treatment. In their own 150 cases they record two of complete and four of presumed complete remission; by this is implied that blood and bone marrow were normal and clinical signs no longer present, and the diagnosis of acute leukaemia could no longer be made; another seven patients showed partial remission. Of the six patients with complete remission four had only supportive treatment and survived 46, 30, 34, and 28 weeks respectively; one had nitrogen mustard and lasted 38 weeks, one had $_{32}P$ and lived 17 weeks; the patients with partial remissions survived from 12 to 52 weeks.

Facts are also given about the initial symptoms, the physical findings, the blood and marrow pictures, the blood groups, the results of blood-clotting tests and serological tests. Brief references to the bone involvement in sixteen of their cases are made. Although chronic lymphatic leukaemia is said to be commoner among Jewish patients, acute leukaemia shows no such incidence. There was no decisive increase in the number of admissions to the Memorial Hospital over the years 1926 to 1948 that could not be explained by greater interest in treatment and better diagnosis. Brief case records are given of the thirteen cases with remission and of two patients who survived for an unusually long period without any apparent remission.

M. C. G. Israëls

Red Cell Survival Studies in Normal and Leukaemic Subjects. A Latent Haemolytic Syndrome in Leukaemia with Splenomegaly—the Nature of Anaemia in Leukaemia—effect of Splenectomy. (In English.) BERLIN, R. (1951). *Acta med. scand.* Suppl. 252, **139**, 1. 59 figs, bibl.

Haemopoiesis and Siderosis in the Foetus and Newborn. LANGLEY, F. A. (1951). *Arch. Dis. Childh.*, **26**, 64. 4 figs, 44 refs.

A study was made of the amount of erythropoietic tissue and stainable free iron in the organs of the newborn; the results are described, and form the basis of a reconsideration of the changes in the haematopoietic system at birth. Sections of liver, spleen, and kidney were examined from 92 infants, eighteen of whom were stillborn and 74 ranging in age at death from 10 minutes to 7 months. Of the total, 48 were premature and 44 full-term infants. In none was there evidence of haemolytic disease. The limits of prematurity were 38 weeks' gestation and 48 cm. crown-heel measurement. Among stillborn infants the mean content of haematopoietic foci in the liver was clearly greater in the premature group, though no relationship emerged between amount of haematopoietic tissue and the degree of prematurity. Prenatal recession of erythrocyte production occurred in the 38th and 40th weeks.

In the liveborn group a process of post-natal recession began at 2 to 3 days of age and was complete about the eighth day in full-term infants, but was prolonged by prematurity, according to its degree, up to 38 days duration. This period was considerably less than the number of days of prematurity. Postnatal recession differs from the foetal type, and closely follows a logarithmic law. Visible iron pigment was present in the liver in two-thirds of the cases, including half of the stillborn group. No correlation was found between siderosis and prematurity, but siderosis and haematopoiesis occurred more often apart than together. Siderosis was rarely present between the first and third days, increased to the fourth week, and decreased after 3 months. Incidence in the spleen followed that in the liver; there was a positive reaction to iron in only two instances in the kidney.

The post-natal fall in the erythrocyte count in the

second week of life and the physiological jaundice of the newborn have been attributed to haemolysis. The author regards the former change as reflecting the diminishing haematopoietic function of the liver; the iron supplied for this activity accumulates as less is used, and some is deposited in the spleen. The latter condition may result from immaturity of liver function.

More extensive knowledge of such factors as changes in blood volume at birth, rate of production of both liver and marrow erythrocytes and their attributes, is required before the part, if any, played by haemolysis can be assessed.

V. Reade

Maternal States in Relation to Congenital Malformations. CARTER, C. O. (1950). *J. Obstet. Gynaec. Brit. Emp.*, **57,** 897. 15 refs.

A study was made of the antenatal records of 14,813 mothers who had attended since the early months of pregnancy at Queen Charlotte's Hospital, London, were confined in the hospital after the 28th week of pregnancy, and were delivered between January, 1943, and July, 1949, for evidence of factors tending to increase the rate of congenital malformations in their babies. The number of malformed children was 219, an incidence of 1·47 (±0·10) per 100 live and still births. Where two malformations were present the child was listed under the major malformation, where more than two were present under " multiple ".

The possible factors investigated were maternal age, birth order, maternal infections, maternal morbidity, and previous obstetrical incidents. Increasing maternal age was associated with increased incidence of mongolism, but if mongols were excluded it had no effect on the incidence of malformations as a whole. Birth order had no effect on the incidence of malformations as a whole, whether mongols were included or not, or on the incidence of the individual malformations of mongolism, anencephaly, or spina bifida. There was no significant seasonal variation in the incidence of malformations during the 6 years of the survey, or variation in monthly totals, and so no evidence of an appreciable effect of the two common groups of virus infections, coryza, and influenza.

Of 1,560 cases of maternal morbidity early in pregnancy, 22 resulted in malformed children (1·41 (±0·30) per 100 live and still births.) Of the commoner abnormal maternal states, uterine retroversion, uterine myomata, mitral stenosis, and vaginal haemorrhage were not associated with an increased incidence of malformed infants; seven were born to 658 mothers with retroversion, an incidence of 1·05 (±0·40) per cent.; two to 206 mothers with uterine fibroids (0·98 per cent.); two to 168 mothers with mitral stenosis, and eight to 294 mothers who had vaginal haemorrhage of any degree in the first 16 weeks of foetal life, an incidence of 2·72 ±0·95) per cent. None of these percentages is significantly different from the general incidence. All 23 mothers with severe vaginal haemorrhage gave birth to normal children. Of the less common maternal conditions in smaller numbers of cases, placenta praevia (52), virus infections in the first 16 weeks of foetal life (52), diarrhoea and vomiting (9), pulmonary tuberculosis (33), bronchiectasis (13), ovarian cyst (31), enlarged thyroid (17), and diabetes (8) were not associated with an increased incidence of malformed infants.

Of the events in the obstetrical history, previous abortions were not followed by an appreciable rise in the rate of malformations, while previous malformations of all types taken together were not followed by any considerable rise in the incidence of malformations as a whole. Certain specific malformations, however, tended to be repeated with specific frequency in subsequent pregnancies.

John D. Hay

Vitamin B_{12}, a Factor in Prevention of Hydrocephalus in Infant Rats. O'DELL, B. L., WHITLEY, J. R., and HOGAN, A. G. (1951). *Proc. Soc. exp. Biol., N.Y.*, **76,** 349. 1 fig., 6 refs.

It was found some years ago that feeding rats on a synthetic diet containing casein as the only source of protein causes hydrocephalus in about 2 per cent. of the offspring. The present authors have shown earlier that this is due to a nutritional deficiency and that it could be prevented by the addition of aqueous liver extract, and in some instances also by addition of folic acid, to the diet. They observed an increase in the incidence of hydrocephalus up to 20 per cent. in the offspring when a folic acid inhibitor, crude methylpteroylglutamic acid (methyl P.G.A.), was added to a diet that contained soy-bean oil meal as the source of protein. The type of diet consumed during the pre-experimental period had a marked effect on the incidence, and this suggests that the protective factor may be stored in the mother rats.

To test the importance of vitamin B_{12} three groups of rats were used. Group I was maintained on a basal diet free of vitamin B_{12} for at least 3 months in order to deplete them of the protective factor. Afterwards, during the experimental period, the animals received the same diet to which crude methyl P.G.A. was added. Group II was not depleted but received the same diet as Group I. Group III was also not depleted, but received vitamin-B_{12} concentrate in addition to methyl P.G.A. in the diet. The incidence of hydrocephalus was 23 per cent. in the offspring of Group I, 15 per cent. in Group II. The same difference was seen in the second litters, but after the third litter there was very little difference. In Group III (vitamin B_{12} added) no hydrocephalus was observed among the 1,261 offspring. In order to verify that vitamin B_{12} was responsible for this protection, and not some other factor in the concentrate, crystalline vitamin B_{12} was injected into five female rats that had produced at least three consecutive litters with hydrocephalus. No hydrocephalic offspring were observed. Efforts were made to determine when vitamin B_{12} had to be injected in order to prevent the abnormality; the damage occurred after the seventh day of gestation, probably about the twelfth to fourteenth days.

Addition of folic acid in the absence of vitamin B_{12} could not prevent the abnormality.

Z. A. Leitner

A Study of the Association of Factors of Pregnancy and Parturition with the Development of Cerebral Palsy. A Preliminary Report. LILIENFELD, A. M., and PARKHURST, E. (1951). *Amer. J. Hyg.*, **53**, 262. 2 figs, 14 refs.

An attempt has been made to elucidate by the statistical method the relation of complications of pregnancy and parturition, of operative procedures, prematurity, and birth-order distribution to the development of cerebral palsy. A series of 561 cases of cerebral palsy in babies born between 1940 and 1947 was collected from the case index of the Crippled Children's Program maintained by the New York State Department of Health. Since 1940, physicians have entered on the birth certificates various factors relating to pregnancy and parturition: these are the sources of the information used in this study.

The percentage of each of the complications of pregnancy and parturition (eleven were used) was significantly higher in the cerebral-palsy group than in the surviving living-birth group. That there are no such complications in the history of a large percentage (62·2) of cerebral-palsy cases is explained by Rh incompatibility, post-natal factors, unrecorded or misdiagnosed information, and hereditary factors. It is of interest to note that such complications as placenta praevia and premature separation of the placenta, which are more prone to produce anoxia, are more closely associated with cerebral palsy than are such conditions as dystocia and abnormalities of the bony pelvis, which are more prone to produce mechanical trauma. Whereas the percentage distribution of complications was similar in the cerebral-palsy group and the infant-loss group (stillbirth and neonatal deaths), there were three exceptions: premature separation of the placenta, complications of the umbilical cord, and toxaemia of pregnancy. The two former are more often associated with infant loss, probably because they do more lethal damage; the latter, which probably produces sublethal effects, is more often associated with cerebral palsy. Although the frequency of various operative procedures is higher among the cerebral-palsy cases than in the total birth group, consideration of the associations of the no-complication group shows the differences to diminish to some extent. No final conclusions could be drawn from that factor group.

Prematurity and cerebral palsy have been associated by many workers, and this is again confirmed. It is shown that this association is independent of whether or not the prematurity is the result of a complication of pregnancy or parturition. The order of birth and age of the mother in the cerebral-palsy group showed a similarity to the pattern of the association existing with the combined infant loss.

From this study there appears to be a definite relationship of various conditions associated with childbearing to the subsequent development of cerebral palsy, although the source of the material imposes certain limitations on its interpretation. As further elucidation of this very important subject might result in programmes that effected a diminution in " reproductive wastage " a new scheme was introduced in New York State in January, 1950, for the mandatory reporting of cerebral palsy. It is hoped that this will provide material for a more extensive and more detailed analysis.

David Morris

The Assessment of Results in the Conservative Treatment of Cerebral Palsy. CROSLAND, J. H. (1951). *Arch. Dis. Childh.*, **26**, 92. 1 ref.

Mental Achievement of Congenitally Hypothyroid Children. A Follow-up Study of Twenty Cases. TOPPER, A. (1951). *Amer. J. Dis. Child.*, **81**, 233. 10 figs, 43 refs.

The author studied twenty cretins aged from 2 to 35 years. Thyroid treatment had been begun at from 2 months to 12 years of age, and had been satisfactory as shown by clinical effect, *x*-ray estimation of bone maturation, and blood cholesterol level in all but two including that untreated up to 12 years of age. Good correlation was found between the treatment and physical, skeletal, and sexual development in all the children. Of the eighteen cretins adequately treated, seven had intelligence quotients below 70, two from 80 to 89, seven from 90 to 110, and two over 110. Electroencephalograms were recorded satisfactorily from six mentally defective cretins and from six mentally normal cretins. The latter gave normal records, but five of the defectives showed diffuse cerebral dysfunction. The author believes that electroencephalographic investigation in infancy in cretins can be used to foretell the future intelligence level.

G. de M. Rudolf

Congenital Anomalies of the Hand. BARSKY, A. J. (1951). *J. Bone Jt. Surg.*, **33-A**, 35. 42 figs, bibl.

The author describes 63 cases of varying congenital deformity of the hand. Almost half the cases showed syndactylism, often in combination with other deformities, and the operation of choice was the reflection of a flap to form a base for the web space with a free graft to the adjacent sides of the fingers. Polydactylism presents few problems, but the need for care in amputation is stressed, and it is important that tendons and muscles attached to the supernumerary digit be transplanted to the next finger. Annular grooves rarely occur as an isolated deformity; when deep they may need excision and plastic repair to prevent the development of oedema.

A number of less common deformities are described, including cleft hand, congenital amputations, and defects of the forearm. Of these patients, three had a family history of congenital limb deformity, and a discussion of the agents which give rise to congenital defects suggests that it is the age of the foetus rather than the nature of the stimulus which is the important factor in determining the type of deformity produced.

Peter Ring

Latent Pyloric Stenosis. (Stenosi pilorica latente). BIEBER, A. (1951). *Riv. Clin. pediat.*, **2**, 93. 12 figs, 6 refs.

A form of pyloric stenosis was described by Durand in 1949 which did not present any characteristic clinical

picture but only a diminished, rather inconstant speed of evacuation from the stomach; it was demonstrable radiologically. The present author describes 6 cases of another form of stenosis which he terms latent pyloric stenosis. Anorexia is the main feature in the history, no matter whether the child is breast-fed or bottle-fed, premature or full-term, weak or strong at birth. In these cases feeding regimens had been quite satisfactory, so that the lack of interest could not be attributed to faulty feeding. Vomiting occurs; it is irregular, scanty in volume, and never violent as in pylorospasm or stenosis. It tends to appear early in life, perhaps at the end of the first week, and it may occur only at long intervals—weeks or even months. It usually occurs immediately after a feed. These characteristics distinguish it clearly from the vomiting of stenosis or spasm. Another feature of the condition is the weight curve, since this remains level or very slightly rises, unlike that occurring in the other two conditions. Constipation is not common, though stools tend to be small and dry. Peristalsis and a pyloric tumour have not been observed.

Radiological examination shows that an opaque meal begins to pass through the pylorus at the usual time but the stomach takes much longer to empty, the time varying from 4½ hours (slightly above normal) to 12 hours. The appearance of the opaque stream while in the pylorus is normal. Unlike the findings in stenosis, the stomach is of normal size and the movements appear to be quite regular. Treatment is largely dietetic. Antispasmodics are ineffective, and operation is not indicated in the absence of a tumour. Parenteral methods may be needed if there is severe wasting or feeding by mouth does not give sufficient volume. The guiding principle is to introduce small amounts of food, aiming first at giving sufficient to maintain life, then gradually increasing the quantity as toleration increases. On the whole, intervals of 4 to 5 hours between feeds are necessary, as otherwise the stomach never empties properly.

It is important to recognize this form of stenosis, since an untreated child may grow up extremely stunted from chronic underfeeding. *J. G. Jamieson*

Pyloric Stenosis in Twins. POWELL, B. W., and CARTER, C. O. (1951). *Arch. Dis. Childh.*, **26,** 45. 20 refs.

Pyloric Stenosis in Four First Cousins. CARTER, C. O., and SAVAGE, T. R. (1951). *Arch. Dis. Childh.*, **26,** 50. 1 fig., 4 refs.

Diabetes and Pregnancy: with Special Reference to the Prediabetic State. MOSS, J. M., and MULHOLLAND, H. B. (1951). *Ann. intern. Med.*, **34,** 678. 3 figs, bibl.

From among a series of five hundred diabetic women seen over a period of 20 years, records of 450 pregnancies, occurring in 72 of them either in the diabetic or prediabetic state, were available: these records form the basis of the study reported. The statistics found are in general agreement with those of most other writers on the subject: thus in the diabetic group the over-all foetal mortality rate was 54·7 per cent. and bore little relation to the severity of the maternal diabetes; the usual high incidence of large babies (weight exceeding 10 lb. (4·5 kg.)), late pregnancy toxaemia, stillbirths, neonatal deaths, and spontaneous abortions was found; the occurrence of these complications in the 30 years preceding the onset of clinical diabetes, but particularly in the last 5 of those years, was once again observed and gave added confirmation to the existence of the " pre-diabetic state ".

The authors discuss the relation of so-called benign renal glycosuria in pregnancy to latent or mild diabetes and point out the difficulties of making the correct diagnosis. Since the outcome of the pregnancy may depend to some extent upon this, they feel that a postprandial urine specimen should be tested for reducing substances at each ante-partum visit while all patients with glycosuria, obesity, and a family history of diabetes, or who have previously had any of the complications of pregnancy common to diabetics and pre-diabetics, should have a glucose tolerance test. They state that if the fasting blood sugar level exceeds 120 mg. per 100 ml. of blood, or the level at one hour exceeds 180 mg. per 100 ml., or that at 3 hours is still above 120 mg. per 100 ml., the patient should be classed and treated as a diabetic. They treated a series of six pregnant diabetics with stilboestrol and progesterone as advocated by White (*Treatment of Diabetes Mellitus*, 8th. edit., 1947, Philadelphia) without any foetal death; none of the babies was large and no mother required Caesarean section. They admit that no conclusions can be drawn from so small a series, but point out that in these patients' 26 previous pregnancies the foetal mortality rate was 27 per cent. and that only 38·5 per cent. of these pregnancies were entirely normal. *G. I. M. Swyer*

Gastric Tumours Associated with Pernicious Anaemia. (Les tumeurs gastriques au cours de l'anémie de Biermer). DEBRAY, C., ROBERT-BENON, and DEBRAY, J. (1951). *Sem. Hôp. Paris*, **27,** 1641. 9 figs, 35 refs.

In eight of 41 patients with pernicious anaemia gastric neoplasms were found; of these, five were benign polypi, two carcinoma, and one a polyp which apparently became malignant. Benign polypi were found in three women and two men aged from 54 to 74 years. Four cases detected by gastroscopy showed polypi which were very small, well defined, rounded, and, with one exception, sessile; they were present in the pyloric antrum and body of the stomach. Gastric carcinomata developed in two patients 15 months and 4 years respectively after the onset of pernicious anaemia.

The last case is of special interest. A 71-year-old man who developed pernicious anaemia showed no abnormality on gastroscopy other than atrophic gastritis, but 2 years later a small sessile polyp was found on the greater curvature. After 3 more years a marked change was detected in the tumour, with enlargement, bleeding, and an irregular surface. The patient was unfit for operation but, over the next 18 months until his death, the development of the growth to a large malignant mass

was observed by the gastroscope. A necropsy was not carried out.

In a long discussion of the relationship between pernicious anaemia and gastric tumours the literature is reviewed to show that the incidence of benign and malignant gastric neoplasms is considerably higher in people with pernicious anaemia. The authors find gastroscopic more valuable than radiological investigation in detecting polypi, and they believe that patients with pernicious anaemia should be examined yearly for gastric tumours and more often if polypi are present.

Peter Story

Statistical Studies of Heart Disease. IX. Race and Sex Differences in the Trend of Mortality from the Major Cardiovascular-Renal Diseases. MORIYAMA, I. M., and WOOLSEY, T. D. (1951). *Publ. Hlth. Rep., Wash.*, **66,** 355. 4 figs, 9 refs.

Mortality in the U.S. from " major cardiovascular-renal diseases ", comprising all forms of nephritis, infections of the heart (excluding syphilis and acute rheumatic fever), functional heart disease, chronic myocarditis, coronary artery disease, and intracranial lesions of vascular origin, has been analysed according to age, sex, and race; the yearly changes in the period 1920 to 1948 have been determined. Among white females the death rate has fallen over this period, but to a lesser degree in each successive 10-year age group, up to 84 years. Above this age it has increased, probably owing to " senility " being replaced as a cause of death on death certificates by " cardiovascular disease ". For non-whites, the death rate is steady or decreasing in all age groups, the trends being similar for males and females. Among white males there is a marked increase in death rate in the age groups 35 to 64 years during this period, the other age groups showing a fall or remaining steady.

In the youngest age group (25 to 34) the death rate for non-whites is much greater than for whites. The difference becomes progressively less in the higher age groups, and in the age group 65 to 74 the rates are equal. Above this age the death rate for whites is greater than for non-whites. This reversal is probably not significant, as a similar change is found when deaths from all causes are considered. It may be due to under-registration of deaths and age inaccuracies in death certificates for negroes.

In 1920 the male and female death rates were about the same at all ages, but in 1947 the male rate was about double the female at all ages. The increase in the death rate for white males is statistically significant and does not occur when death rates from all causes are considered. The difference in trends for males and females is most marked in diseases of the heart and chronic nephritis.

M. Lubran

The Outcome of Mental Breakdown in Old Age. POST, F. (1951). *Brit. med. J.*, **1,** 436. 1 fig., 11 refs.

An analysis was made of the records of all patients over 60 admitted to a mental observation unit in South London from February to May, 1946. Of 454 persons admitted, 105 men and 121 women were over 60, but twelve patients with primarily physical disease were excluded from the study. The remainder (214) were divided into two broad diagnostic categories: 26 men and thirty women were suffering from essentially " functional " (schizophrenic or affective) psychosis, while 69 males and 89 females exhibited organic mental symptoms.

A table shows the numbers of each category dying under hospital care; discharged; and remaining in hospital. The follow-up over $3\frac{1}{2}$ years revealed striking differences in discharge and death rates of the two groups. Only a small proportion of old people suffering from mental breakdown occupy hospital beds for prolonged periods, those remaining after a year being about one-quarter. The results of a follow-up from records are given and a comparison is made with the findings of Clow (*N. Y. St. J. Med.*, 1948, **48,** 2357) and of Camargo and Preston (*Amer. J. Psychiat.*, 1945, **102,** 168). A table of age distribution and mortality is given.

To assess the importance of social factors in causation and outcome of mental breakdown in this group, 190 patients with no previous psychiatric breakdown were reviewed. A table shows the type of illness, marital status, and degree of social integration of these 190; the results are compared with those of Rowntree (*Old People*, London, 1947).

Although only 3 per cent. of the elderly population is estimated to live in institutions and homes for the aged, 17·4 per cent. of these patients had passed through public assistance institutions. This point is discussed. Persons admitted from institutions or from a lonely existence are older as a group than patients who had remained socially integrated.

The author concludes that the degree of social integration appears to have little influence on the outcome of mental breakdown.

John C. Kenna

Proper Use of the Hospital in Treatment of the Aged Sick. LORD AMULREE, EXTON-SMITH, A. N., and CROCKETT, G. S. (1951). *Lancet*, **1,** 123. 12 refs.

The treatment of the aged sick is one of the greater problems of the hospital and specialist services under the National Health Service in Great Britain. The final disposal of a group of aged sick patients in St. Pancras Hospital, before the initiation of a new service, based on the hospital, to provide better facilities for the care of such persons, is analysed and compared with the disposal of a series of patients admitted since the service was started. The two series are admittedly not strictly similar, but the general conclusion is drawn that much can be accomplished given a certain enthusiasm in the medical and nursing attendants. A special point is made of the value of domiciliary visits in the first instance before admission, and a plea is made for the greater encouragement of activity in aged patients while in hospital, and the provision of hostel accommodation and other facilities to enable a quick turnover of the hospital population of aged sick.

Scott Thomson

Mass Radiography Findings in the Northamptonshire Boot and Shoe Industry, 1945-6. STEWART, A., and HUGHES, J. P. W. (1951). *Brit. med. J.*, **1**, 899. 13 refs.

This important paper analyses mass radiography (M.R.) findings in the boot and shoe industry in comparison with those obtained in other industries (engineering, clothing, leather dressing) and in professional occupations. The population under review has been divided into two age groups for each sex: under 35, and 35 and over. The average " coverage " (the percentage of the total number of employees attending the surveys) has been round about 70 per cent. [This implies that a significant number of tuberculous persons may have been missed (Bradbury, *Lancet*, 1948, **2**, 293).] The authors finding that there is a relatively high incidence of active and of quiescent tuberculosis among factory operatives in the boot and shoe industry, the incidence varying directly with the size of factory, and their conclusion that this industry attracts men of poor physique are derived from a detailed statistical analysis of all the relevant factors, obtained from the examination of 18,660 employees of boot and shoe factories and of 23,738 subjects drawn from other occupations.

The study [which repays reading in its original form] includes a wide range of interesting and significant findings: the epidemiological significance of " quiescent " and of previously notified cases is brilliantly demonstrated. It could be established that " the incidence of active tuberculosis was a function of factory size ". The importance of droplet infection amongst fellow workers could be unequivocally proven, revealing the dependence of new infections on " room distribution ". The preference of tuberculous subjects for the lighter type of work within the industry could be shown. This paper not only reveals statistically significant facts about the incidence and the age and sex distribution of tuberculosis in boot and shoe operatives as compared with other workers, but it also contains new and most valuable suggestions as to how an M.R. service could be more usefully employed: in addition to simple case-finding, M.R. could and should make an important contribution to epidemiology and to environmental research with the aid of specially trained workers. M.R. could, in fact, substantially further our knowledge of " social pathology ".

[A more economical and systematically planned use of M.R. and its direction towards specific targets rather than towards " routine surveys " have been suggested elsewhere (Hoffstaedt, *Lancet*, 1947, **2**, 955; *ibid.*, 1950, **2**, 756).]

E. G. W. Hoffstaedt

The Role of Periodic Examination in the Prevention of Coalworker's Pneumoconiosis. COCHRANE, A. L., FLETCHER, C. M., GILSON, J. C., and HUGH-JONES, P. (1951). *Brit. J. industr. Med.*, **8**, 53. 1 fig., 32 refs.

Since complete suppression of all airborne dust in mines is impracticable and there is no reliable method of determining the safe level, the authors believe that periodic medical examination of coal-miners is the method that would most quickly and economically lead to the prevention of disabling pneumoconiosis. They review the evidence for the beliefs that early radiological change can be detected before serious disablement occurs and that men with early radiological change will not develop progressive massive fibrosis and consequent disability if they are removed from exposure to dangerous dust. They envisage that, as a result of periodic examinations, the only men who would be advised to leave the industry or to change their occupation to one involving less dust exposure would be those with early simple pneumoconiosis that was found to be advancing on serial *x*-ray examination, especially if their exposure had been short, and those who were actually disabled by the disease.

The authors also consider that such periodic examinations, combined with measurements in the mines, would provide a means of defining safe dust levels in different mines. They suggest that a pilot scheme should be started, preferably in South Wales, and then be extended on a voluntary basis to mines representative of various areas in different coalfields.

H. E. Harding

Medical Significance of the Census. LOGAN, W. P. D. (1951). *Brit. med. J.*, **1**, 720. 1 fig., 12 refs.

The census is necessary for the calculation of those indices used in measuring the state of the public health. Knowledge of the sex and age distribution of the population is essential for correcting the crude indices and revealing the true changes with time. In addition, the construction of national life tables and calculation of the expectation of life can be carried out accurately only from the census figures. Occupational mortality can be deduced by analysing the occupations of the living, as returned in the census, and the occupations of those dying in 1950-52, as given at registration of death. By analysing deaths of married women according to the occupation of their husbands, it is hoped to distinguish between socio-economic and occupational factors. Calculation of infant mortality according to the occupation and social class of the father will also be made. There will be no attempt to enumerate the sick, as previous experience has shown the unreliability of the results.

M. Lubran

Genital Haemorrhage in the Menopause, with Special Reference to Cystic Glandular Hyperplasia. (Genitale Blutungen in der Menopause mit besonderer Berucksichtigung der glandular-cystischen Hyperplasie.) WAGNER, H. (1950). *Arch. Gynäk.*, **177**, 460. 3 figs, 21 refs.

Post-menopausal bleeding is regarded as being due to a malignant condition until it is proved after investigation to be due to some other cause. The recorded frequency of malignancy as a cause of post-menopausal bleeding varies from 27·8 to 78 per cent. The difference

is due to the selection of cases: sometimes patients with amenorrhoea lasting for 6 months are included while other observers require a longer period of amenorrhoea; again some workers include all cases of bleeding, others exclude such cases as those of varicose veins of the vulva or vagina and senile endometritis where no tissue is available for histological examination.

The present author studied patients who had had amenorrhoea for at least 3 years and excluded cases where histological examination of tissue was not possible. In 240 of 253 cases the bleeding was uterine in origin, while in thirteen cases the cause was extra-uterine (vulval and vaginal carcinoma, four cases; vaginal ulceration, nine cases). Of the 240 cases of uterine bleeding, 103 were due to a malignant condition. Carcinoma of the cervix was present in 37 patients, twenty of whom were under 60 years of age, and carcinoma of the body of the uterus in 66 patients, 28 of whom were under 60. Two patients had sarcoma of the uterus.

Cervical polyp was the commonest non-malignant cause of uterine bleeding, 38 cases (15·8 per cent.). Atrophic endometrium was found in 24 cases and cystic glandular hyperplasia in 23 cases. These 23 cases are discussed in detail. The author refers to the work of Novak and agrees with him that the condition is a pre-cancerous one; details of two cases are given: in one, carcinomatous change was found 4 months after cystic glandular hyperplasia, while in the other the two conditions were found together. Of this group of patients, one had a granulosa-celled tumour, eight had had ovarian hormone therapy, while in the remaining fourteen no obvious cause was found to account for the changes in the endometrium. Histological examination showed a striking difference in the two groups. When hormone therapy had been given the endometrium was thick and contained only isolated cystic glands, while in the group with bleeding where no obvious cause was found, the endometrium was thinner and had islands of glands with a varying degree of dilatation and branching. This difference in the two groups is, in the author's view, significant, and he suggests that the cystic glandular hyperplasia of the " unknown-cause group " is a simple new growth, which, as already mentioned, may become malignant.

Gladys Dodds

The Psychopathology of Ovulation and Menstruation. (Die Psychopathologie der Ovulation und Menstruation.) HUTTER, A. (1950). *Schweiz. Arch. Neurol. Psychiat.*, **66,** 159. 2 figs, 4 refs.

The relation between ovulation, menstruation, and affective disorders is generally recognized, but insufficient attention has been paid to the association of menstrual changes with the outbreak and the course of other psychoses. The author describes a case of schizophrenia in which acute paranoid and catatonic disorders were always precipitated and aggravated by menses. The psychopathology of this group of psychoses, which the author calls " menstrual psychoses ", is discussed.

J. T. Leyberg

Statistical Evaluation of Morning Temperature Curves for Recognition of the Laws of the Human Menstrual Cycle. (Statistische Auswertung von Morgentemperaturkurven zur Erkennung von Gesetzmässigkeiten im weiblichen Menstruationscyclus.) HUBERT, W. (1950). *Arch. Gynäk.*, **177,** 473. 4 figs, 23 refs.

A statistical evaluation is made of the waking temperature curves during 242 menstrual cycles in inmates of a sanatorium after rejection of all monophasic temperature graphs, which amounted to 50 per cent. of the total material. There were great individual variations in the length of the whole menstrual cycle and in that of its two phases. There was close correlation between the length of the whole cycle and that of the follicular phase, but there was no correlation between the duration of the whole cycle and that of its luteal phase. The author distinguishes four types of menstrual cycle: (1) cycles of " normal " duration of from 27 to 29 days, the two phases being of approximately equal length; (2) moderately shortened or moderately lengthened cycles, usually at the expense of the follicular phase; (3) considerably shortened cycles with equal shortening of both phases (" miniature cycles "); (4) considerably lengthened cycles with comparatively constant length of the luteal phase.

[It would appear doubtful whether the findings in women suffering from pulmonary tuberculosis, described as " mild " by the author but nevertheless necessitating treatment in a sanatorium, provide a suitable basis for general conclusions about the menstrual cycle.]

N. Alders

The Basal Temperature Curve in the Diagnosis of Menstrual Disturbances. (Der Wert der Basaltemperatur für die Diagnose der Menstruationsstörungen.) PLOTZ, J. (1950). *Arch. Gynäk.*, **177,** 521. 27 figs, bibl.

This is an extensive study of waking temperature curves during normal and abnormal menstrual cycles, obtained from 200 women; 10 to 12 cycles were recorded in some cases, and a great number of endometrial biopsy examinations were performed and results correlated with the temperature curves.

The author describes an " intermediate type " (*Zwischentyp*) of menstrual cycle in which there is no biphasic temperature curve, but small, irregular rises of temperature on 1 or 2 days before the onset of bleeding, which does not last longer than 3 days and is scanty in amount. Biopsy examination of the bleeding endometrium reveals a predominantly proliferative picture, while some of the glands show secretory changes. [This would appear to correspond to Hamblen's " mixed endometrium ".]

If the cycle is shortened it is either anovulatory, or of the " intermediate type ", or the luteal phase has been cut short. The reason for a lengthened cycle is either persistence of the follicle, " intermediate type ", a " resting of the ovary " after the last menstrual bleeding before a new follicle begins to ripen, or interposition of one or more sub-threshold cycles. Hypomenorrhoea is due either to anovulatory bleeding, to " intermediate

type " cycle, or to shedding of only the superficial parts of the (secretory) endometrium. Irregular shedding of the endometrium is described in detail. Attention is drawn to the fact that a biphasic temperature curve associated with secondary amenorrhoea suggests refractory endometrium or tuberculous endometritis.

The author concludes that careful recording of the waking temperatures will, in 90 per cent. of cases, allow of interpretation and classification of menstrual anomalies.

[Study of this paper, with its numerous curves and photomicrographs, would be worth while to those interested in this subject.]

N. Alders

Sperm Survival in Women. Motile Sperm in the Fundus and Tubes of Surgical Cases. RUBENSTEIN, B. B., STRAUSS, H., LAZARUS, M. L., and HANKIN, H. (1951). *Fertil. and Steril.*, **2**, 15. 9 refs.

This investigation is concerned with the duration of sperm survival in the female genital tract. Three volunteers agreed to have the uterus washed out with saline after coitus: in five trials spermatozoa were found after 36 hours still motile in the uterus or in the tubes, as compared with survival up to 60 hours *in vitro* at room temperature for the same specimens. Owing to the occurrence of endometrial infection in two out of the three volunteers the experiments were discontinued. The next step was to spray semen on the cervix of patients about to undergo hysterectomy. The cervix, fundus, and tubes were then separated immediately after operation, opened and washed with saline, and a search made for spermatozoa. The results show that spermatozoa reach the tubes within 30 minutes and may survive up to 50 hours. No correlation was attempted with the phases of the menstrual cycle.

[This extremely interesting work needs to be repeated with a larger number of cases to correlate different types of results during ovulatory and during other phases of the menstrual cycle. One important point not mentioned in the report is whether inquiry was made as to the date of the last coitus before the patient was admitted to hospital; this may invalidate results such as the sperm migration within half-an-hour.]

B. Sandler

Sperm Survival at Estimated Ovulation Time. Comparative Morphology: Relative Male Infertility. COHEN, M. R., and STEIN, I. F. (1951). *Fertil. and Steril.*, **2**, 20. 2 figs, 5 refs.

This paper provides a study of the comparative morphology of spermatozoa surviving in cervical mucus, as compared with the ejaculate specimen. Previous findings which indicated that sperm may survive up to 72 hours in the cervical mucus at ovulation, but that in the pre- and post-menstrual phases their survival is poor are discussed. Motile spermatozoa were recovered from 1 to 58 hours after coitus and were normal in motility and morphology. A remarkable reduction in the percentage of abnormal spermatozoa was noted. The morphology of spermatozoa after coitus was compared with the analysis of the semen previously obtained from that particular individual. The motility up to 58 hours in the cervix showed the sperm to be as active as in the fresh ejaculate. The morphology and quality of the cervical sperm, however, were very much better than in the masturbation specimen. Of 33 marriages where a guarded prognosis had been given because of substandard spermatozoa, conception occurred subsequently in 17·1 per cent.

[These results are at variance with those of Rubenstein and others, 1951 (*see* preceding abstract): the explanation may be the low fertility of the women who were undergoing operation for gynaecological lesions.]

B. Sandler

The Testis. III. Absence of Germ Cells: Sclerosing Tubular Degeneration; " Male Climacteric ". SNIFFEN, R. C., HOWARD, R. P., and SIMMONS, F. A. (1951). *Arch. Path.*, **51**, 293. 8 figs, 25 refs.

Testicular abnormalities in 55 men with oligospermia or azoospermia and hormonal imbalance manifested by increased excretion of urinary gonadotrophins and, in some, low output of urinary 17-ketosteroids and signs of eunuchoidism are described. They could be divided into three categories on the basis of the histological abnormalities observed in the testis. In the first group of nineteen normally developed patients, spermatogenic activity had usually failed completely, but the Sertoli and Leydig cells were intact so that endocrine derangement was minimal. The basement membrane was normal, though the tunica propria usually showed slight thickening. The sensitive germ cells appeared to have been destroyed by some agent operating in the past, but the condition was not the result of maldevelopment of the testis.

The second group of thirty patients showed progressive sclerosis of the seminiferous tubules with hyperplasia of the Leydig cells, many of which were morphologically abnormal. The Sertoli-cell atrophy was always accompanied by thickening of the tunica, and the authors considered that the tubular sclerosis was the result of deposition of collagen fibrils between the inner lamellae of the tunica and not to thickening of the basement membrane. The outer layer of the tunica remained unchanged. These changes were accompanied by increased excretion of urinary gonadotrophins. Output of 17-ketosteroids was variable and seemed to show some correlation with the degree of eunuchoidism and gynaecomastia present.

The authors suggest that the disease probably begins at puberty and that the primary abnormality lies in the Sertoli or Leydig cells, resulting in disturbances in pituitary function. In the third group of six patients, the testis was morphologically normal, but urinary gonadotrophin content was high. The findings were consistent with the so-called " male climacteric ".

A. Ackroyd

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